

ANNALS  
OF  
OTOLOGY, RHINOLOGY  
AND  
LARYNGOLOGY

VOL. 43.

SEPTEMBER, 1934.

No. 3.

LI.

EFFECTS OF IONIZATION ON THE MUCOSA OF FRONTAL  
SINUSES OF DOGS.\*

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Ionization or ionic medication was first mentioned, according to Leduc, by Fabre-Palaprat in 1833. Professor Leduc of Nantes,<sup>1</sup> in an address before the radiologic section of the British Medical Association in 1907, added a fresh impetus which resulted in the general adoption of the method in England. Since that time A. J. Friel,<sup>2</sup> Lewis Jones<sup>3</sup> of London and others principally in England,<sup>4</sup> France<sup>5</sup> and America<sup>6</sup> have advocated this procedure more or less strongly.

While the literature is replete with publications, especially in the last twenty years, one is struck by the fact that now, as in the beginning, reports of good results from treatment by ionization vary from 10 to 85 per cent of cures in otitis media,<sup>7</sup> with a few sporadic good results reported in sinusitis<sup>8</sup> and vasomotor rhinitis.<sup>9</sup>

The rationale is based upon the principle that like electrical charges repel each other. In an electrolyte through which no current is passing the ions are in perpetual movement, but the movement is not in any definite direction. When a current is sent through an electrolyte, the movement becomes orderly, and is directed towards

\*Presented before the annual meeting of the American Laryngological Association at Cleveland, June 7, 1934.

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the poles. Thus by introducing a positive electrode into an electrolyte such as zinc sulphate, the zinc ion carrying a positive charge, and causing a galvanic current to flow through the solution, the zinc ion is theoretically driven into the tissues with which the solution is in contact. The same phenomenon occurs when the negative electrode acts in a negative solution.

That such therapeutic ions as silver, iron, magnesium, adrenalin, lithium, morphin, strychnin, aconite and arsenic may be introduced through the unbroken skin by electrolysis has been demonstrated by Clarke,<sup>10</sup> Pack and others,<sup>11</sup> Laquerriere and Lehmann<sup>12</sup> and Strohl.<sup>13</sup> Lierle and Sage,<sup>14</sup> however, were unable to demonstrate zinc in the mucosa of infected middle ears after ionization with zinc sulphate, either by X-ray, or qualitatively, and not satisfactorily spectrometrically.

Various explanations have been offered as to the action of an electrically charged particle after it invades the tissues. It may be immediately carried away to other parts of the body and exert no local effect whatsoever, or it may lose its charge and become an inert particle, the electrical charge leaving as a nascent ion,<sup>15</sup> this nascent ion attracting oppositely charged bacteria, and thus rendering them inert by causing a coagulation necrosis of the organisms and sterilizing the tissues.<sup>16</sup> Some physicists, however, do not admit the action of nascent ions, but suggest that the effect of the treatment is due to metabolic changes in the tissues consequent upon the galvanic current. Cahill<sup>17</sup> states that the ions of the heavy metals coagulate the tissues and form with some of their constituents more or less insoluble precipitates, that of zinc exercising the greatest coagulative power on fluids containing albumen. It is conjectural whether one or all of these factors may pertain.

In considering the relationship of certain solutions to a mucous membrane we must think of the ionizability of colloidal, as well as metallic solutions, since colloidal suspensions are so generally used in otolaryngologic treatments. Michaelis<sup>18</sup> gives a very clear and concise explanation of this rather abstruse phase of colloid chemistry. He says that—

"The behavior of colloidal ions or 'lazy ions' differs from that of metallic ions. The former are much more sluggish and their electrical charge depends upon external conditions especially the pH. value of the solution. Many substances form a negative colloid under all conditions, and many are amphoteric. The isoelectric point lies at a certain pH.; this occurs either on the acid side as is true of the majority of albuminous bodies, or on the alkaline side as in many metal hydroxides."



Lottermoser<sup>19</sup> states that—

"Some colloidal ions are only colloidally soluble, while others have a wider range of solubility. Thus, for example, Ag I possesses a very slight solubility, but it is perfectly definite owing to the constancy of the solubility product (Ag) (I). A suspension or colloidal solution of Ag I therefore always represents, in addition to the suspended particles of Ag I, some independent Ag and I ions in solution, and neither Ag nor I has the properties of a lazy or colloidal ion. The possibility of a suspension of Ag I exists only when more Ag I is present than is capable of dissolving. This Ag I will be negative if precipitated out of Ag No<sub>3</sub>—by adding an excess of KI, and positive if precipitated out of KI—by adding an excess of Ag No<sub>3</sub>."

This explanation gives us at least a working basis on which to build our hypothesis of the actions of these and other ions which have been used in the treatment of intranasal conditions.

Though many good results have no doubt been obtained, the reasons have been empirical and not based on an analysis of actual tissue changes brought about by ionization, or a consideration of the damage to the membrane which may ensue after ionization.

This experimental study was therefore undertaken to ascertain the immediate reaction of the normal mucosa of the frontal sinuses of dogs when subjected to electrolysis of certain solutions used in routine intranasal medication.

The electrolytes were chosen on account of their general clinical usages with the hope that some practical applications might be aduced from our findings.

#### SOLUTIONS.

1. India ink—Light suspension in 0.1 per cent saline solution.
2. Neosilvol 10 per cent—A colloidal solution containing 20 per cent silver iodide.
3. Argyrol 10 per cent—A colloidal solution containing 20 per cent silver.
4. Zinc sulphate 2 per cent—Aqueous solution.

#### SUBJECTS AND PROCEDURE.

Dogs were chosen because of the easy accessibility of their frontal sinuses. They were prepared by injecting intraperitoneally 6-7 cc. of amytal solution (4.5-5 grs.) about one-half hour before operation. This resulted in a satisfactory anesthesia in which the dog was entirely quiet during the experiment. The skin was reflected upward from over the frontal sinus areas, and with a large gouge sufficient bone was removed in one stroke from the roof of each sinus to furnish a good view of the interiors. A small amount of warm paraffin-

liquid albolene solution, at body temperature so that it solidified as soon as it was dropped upon the small lingula which covered the opening of the nasofrontal duct, was introduced to effectively seal off this outlet. Care was taken not to traumatize in any way the delicate mucosa of the sinuses other than was necessary in uncapping.

Each sinus was filled with the desired solution, the electrode support affixed to the head, and the electrodes, the positive in the left and the negative in the right sinus, inserted into the solution without touching the walls. Pure zinc electrodes were used in the zinc sulphate solution. Platinum electrodes were used in the other solutions because of their electrical inactivity. The galvanic current, turned on slowly until 5 milliamperes was reached, was allowed to remain at that intensity for twenty minutes.

The dog was killed by injecting air into the heart, and the head removed and placed in 10 per cent formalin solution for twenty-four hours, at which time the frontal sinus blocks were removed with a saw, to be decalcified and sectioned. The blocks were embedded in paraffin and serial sections cut.

Dog No. 1, exposed to diathermy, was saved. The sinuses were opened, the mucosa removed and placed in formalin.

Our efforts were directed towards determining the comparative changes in the mucosa under the following conditions:

Dog No. 1. Exposure of the unopened frontal sinuses to 200 milliamperes diathermy for 20 minutes.

Dog No. 2. India ink suspension in 0.1 per cent saline solution, ionized at 5 milliamperes for 20 minutes, in opened frontal sinuses.

Dog No. 3. Zinc sulphate, 2 per cent solution, ionized at 5 milliamperes for 20 minutes in opened frontal sinuses.

Dog No. 4. Neosilvol, 10 per cent solution, ionized at 5 milliamperes for 20 minutes, in opened frontal sinuses.

Dog No. 5. Argyrol, 10 per cent, in right frontal sinus. Neosilvol, 10 per cent, in left for 20 minutes, without diathermy or ionization.

Dog No. 6. Argyrol, 10 per cent solution, ionized at 5 milliamperes for 20 minutes, in opened frontal sinuses.

From the following charts it can be seen that certain constructive as well as desructive changes have taken place, the most marked constructive change common to all the subjects being an engorgement of the capillaries of the subepithelial tissues. In evaluating this reaction we may attribute it largely to the passage of the electric cur-

## CHART 1.—GROSS FINDINGS AFTER IONIZATION.

Dog 1	Right and Left	Mucosa flushed; vessels engorged.
Dog 2	Right and Left	Thin film of India ink covering mucosa. Color and condition of mucosa itself could not be determined.
Dog 3	Right	Solution moderately murky. Flocculent precipitate of apparently coagulated red blood cells on surface and upon mucosa in places. Mucosa extremely flushed, vessels engorged. Heavy black precipitate upon electrode.
	Left	Solution clean. Mucosa unchanged; some vessels moderately engorged. No precipitate upon electrode.
Dog 4	Right	Mucosa covered with glairy film, large numbers of small bubbles, especially on septal wall, less so on posterior wall. Vessels intensely engorged. Large number of similar bubbles about electrode.
	Left	Findings similar to those of right, but much less marked.
Dog 5	Right	Mucosa covered with moderately thick brownish film: Vessels moderately injected.
	Left	Mucosa covered with thin glairy looking film: Vessels not injected.
Dog 6	Right and Left	Mucosa covered with moderately thick brownish film. Vessels markedly injected.

## CHART 2.—MICROSCOPIC FINDINGS AFTER IONIZATION.

	EPITHELIUM	SUBEPITHELIAL TISSUE	BLOOD CAPILLARIES
Dog 1 Rt. and Lt.	Intact—Cilia present.	Extremely thickened by engorged vessels.	Markedly engorged
Dog 2 Rt.	Fragmented carbon in dense bands packed against surface in places; particles are seen penetrating into intercellular spaces, but not completely through. Histocytes in lumen.	No carbon seen. Edema moderate. Many free red blood cells.	Moderately engorged
Lt.	Fragmented carbon particles in intercellular spaces, outlining epithelial cells in places. There is a suggestion of phagocytosis by epithelial cells. Large cells having phagocytosed carbon particles, are free in lumen. Cilia evident in most places.	No carbon seen. Edema moderate cells. No polymorphonuclear leucocytes or histiocytes.	Markedly engorged

CHART 2.—MICROSCOPIC FINDINGS AFTER IONIZATION—Continued.

	EPITHELIUM	SUBEPITHELIAL TISSUE	BLOOD CAPILLARIES
Dog 3 Rt.	Intact in most places, though many fragmented gaps are present through which free red blood cells are seen exuding to surface from subepithelial tissue and forming in large clumps. Also individual red blood cells seen going through intact epithelium to surface.	Extremely edematous. Many free red blood cells. Occasional polymorphonuclear leucocytes.	Moderately engorged. Moderate increase in numbers of polymorphonuclear leucocytes in some.
Dog 3 Lt.	Intact in some places, sloughed and fragmented in others, with marked extravasation of free red blood cells to surface. Many cells show absence of protoplasm, the cell outlines and nuclei remaining intact. Thin bluish gray line on top of cilia and globules of various sizes between and upon cells in several places.	Moderate edema with marked hemorrhage throughout.	Markedly engorged. Few polymorphonuclear leucocytes in some.
Dog 4 Rt.	Intact as a rule. Inter cellular spaces widened, some contain polymorphonuclear leucocytes. Cilia uniformly present throughout.	Few small areas of localized hemorrhage. Many polymorphonuclear leucocytes throughout, but especially around capillaries. Eosinophiles present along floor and lower half of septum.	Moderately engorged. Marked mobilization of polymorphonuclear leucocytes throughout all capillaries.
Lt.	Moderate sloughing in places.	Edema much more marked than right.	
Dog 5 Rt.	Fragmentation extensive, individual cells retaining their cilia. Clumps of polymorphonuclear leucocytes on surface in lumen of sinus.	Polymorphonuclear leucocytes in large numbers throughout. Edema, moderate.	Moderately engorged.

CHART 2.—MICROSCOPIC FINDINGS AFTER IONIZATION—Continued.

	EPITHELIUM	SUBEPITHELIAL TISSUE	BLOOD CAPILLARIES
Dog 5 Lt.	Very little fragmentation. Polymorphonuclear leucocytes going through epithelium in places.	Edematous. Polymorphonuclear leucocytes and free red blood cells marked, much more so than in right. Polymorphonuclear leucocytes also in perivascular spaces of adjacent bone. Large monocytes numerous.	Engorged; some packed with polymorphonuclear leucocytes.
Dog 6 Rt.	Fragmentation and disintegration.	Polymorphonuclear leucocytes in moderate numbers and generally edematous.	Markedly engorged. Polymorphonuclear leucocytes in some about walls.
Lt.	Marked fragmentation and disintegration.	Polymorphonuclear leucocytes in large numbers throughout. Less edematous.	Polymorphonuclear leucocytes packed in some and about walls in others.

rent through the tissues, since it was so evident in the unopened sinuses exposed to diathermy. While a similar engorgement was seen in the subepithelial capillaries of the sinuses exposed to zinc sulphate, there was also present a marked extravasation of the red blood cells into the edematous subepithelial tissues and the lumen of the sinus, and certain more or less extensive destructive changes in the epithelium, decidedly a picture of irritation. These changes, when organization sets in, might lead to permanent thickening of the mucosa by an undue excitation of the connective tissue elements. The engorgement without red blood cell extravasation and the edema of the subepithelial tissues were also seen in the mucosa of the three dogs exposed to the silver colloids, but to a lesser degree. However, in the latter dogs, there was, in addition to the erythrocytic reaction, a very marked mobilization of polymorphonuclear leucocytes within the capillaries and in the subepithelial tissues, and this factor, a stimulation of the phagocytic elements, should be considered decidedly advantageous.

We cannot ascribe this leucocytic reaction solely to the process of ionization, since it was equally as marked in the nonionized sinuses

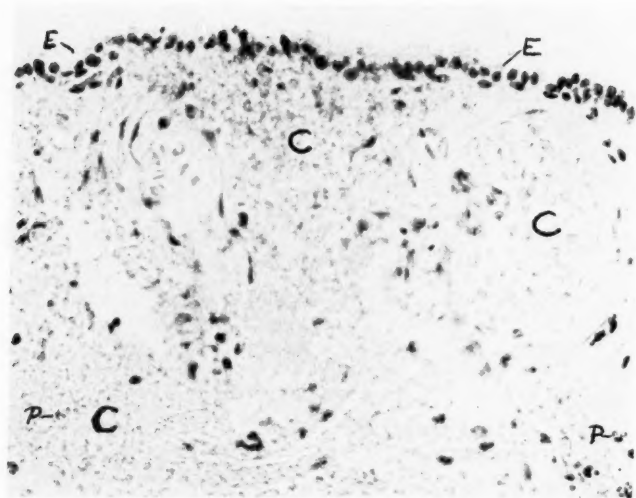


Fig. 1. Mucosa from frontal sinus of dog after exposure to 200 milliamperes diathermic current for 20 minutes. Note extreme engorgement of subepithelial capillaries (C); very few polymorphonuclear leucocytes (P) seen. (E) Intact epithelium showing ciliary line.

as in those which were ionized, though it was slightly more marked in the sinuses exposed to the positive electrodes. This electrode presumably drives the positively charged particles—that is, the silver or the silver-iodide ions, into the tissues, and we might conclude that one of the actions of these ions is to stimulate a localized polymorphonuclear leucocytosis. Argyrol appeared to cause more marked destructive changes in the epithelium than did neosilvol, which caused very little damage in both the ionized and the unionized sinuses. The changes in the mucosa of the septum dividing the two frontal sinuses in the line of direct streaming between the electrodes, were no more pronounced than those in the mucosa of the walls, suggesting that penetration takes place by the diffusion of ions.

The dog ionized with the india ink-sodium chloride suspension was used as a control to ascertain whether the easily seen negatively charged carbon particles would be driven through the intact epithelium with the negative chlorine ions, and if their presence in the mucosa would cause a mobilization of histiocytes.

We found that the carbon particles did penetrate partly through the intact epithelium of the sinus exposed to the negative electrode under electrical stimulation after twenty minutes, in contrast to their

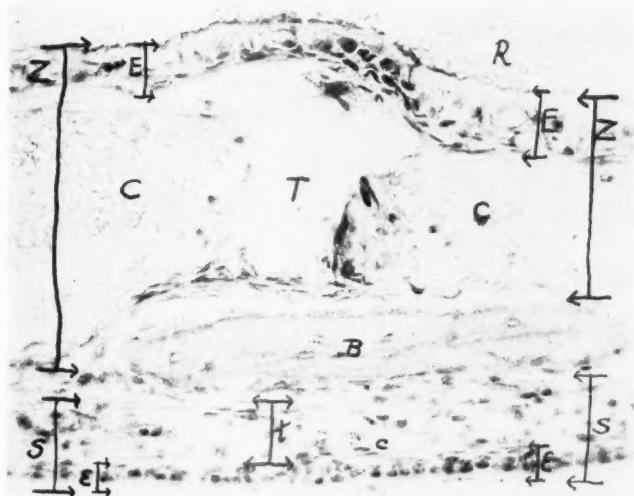


Fig. 2. Mucosa (Z) from frontal sinus of dog after contact with 2 per cent zinc sulphate solution, ionized at 5 milliamperes for 20 minutes, contrasted with mucosa (S) from adjacent accessory frontal sinus not exposed to ionized zinc sulphate solution. E—Epithelium extremely swollen and fragmented, nuclei disintegrated, ciliary line not seen. (e) Normal epithelium, ciliary line plainly seen. T—Subepithelial tissue extremely swollen, containing greatly engorged and dilated capillaries (C). (t) Normal subepithelial tissue containing normal capillaries (c). B—Bone: R—Free red blood cells on surface of epithelium.

inability to penetrate the intact epithelium of the turbinates and antrums of rabbits after six hours without electrical stimulation, as demonstrated in our previous experiments.<sup>20</sup> Histocytes were not seen in the subepithelial tissues, but were numerous in the lumen of the sinuses, and contained phagocytosed carbon particles.

#### SUMMARY.

It is noteworthy that the principal reaction of the mucosa to the silver colloids was cytologic, a localized leucocytosis, and that this was not any more pronounced as a result of ionization, or any less pronounced without ionization. That is to say, an ionizable solution may react spontaneously to the electrical forces of the tissues themselves, when contact is made for a sufficient length of time, and thus enable the constituent ions to penetrate into the mucosa and instigate an appropriate reaction. According to this hypothesis, ionization has no advantage over passive contact within a cavity lined with mucous membrane, in so far as the local tissue reaction is concerned. But



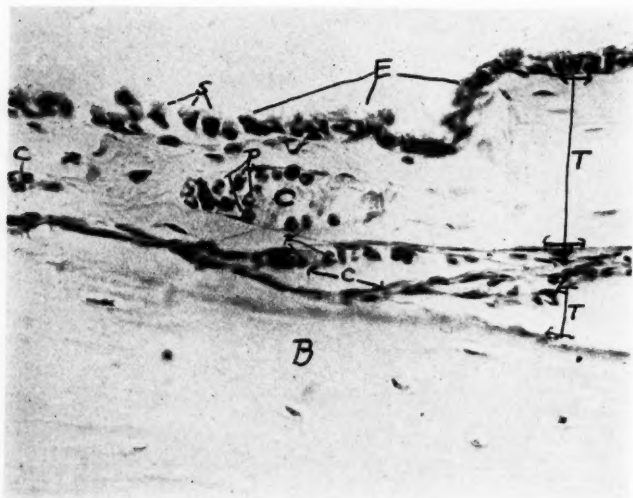


Fig. 3. Mucosa from frontal sinus of dog after contact with 10 per cent Neosilyol solution ionized at 5 milliamperes for 20 minutes. Note engorged capillaries (C) containing large numbers of polymorphonuclear leucocytes (P) in edematous subepithelial tissue (T). E—Epithelium showing some fragmentation, individual cells retaining tufts of cilia (S). B—Bone.

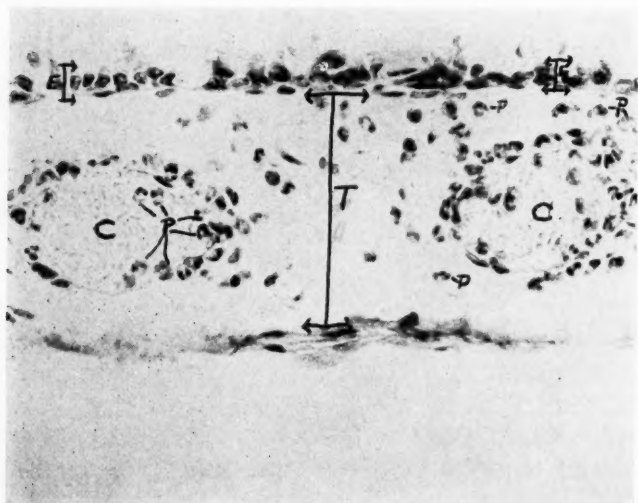


Fig. 4. Mucosa from frontal sinus of dog after contact with 10 per cent argyrol solution for 20 minutes, no ionization or diathermy used. Note engorged capillaries (C) containing large numbers of polymorphonuclear leucocytes (P) which are also free in edematous subepithelial tissue (T). E—Epithelium showing more marked fragmentation than in Fig. 3.

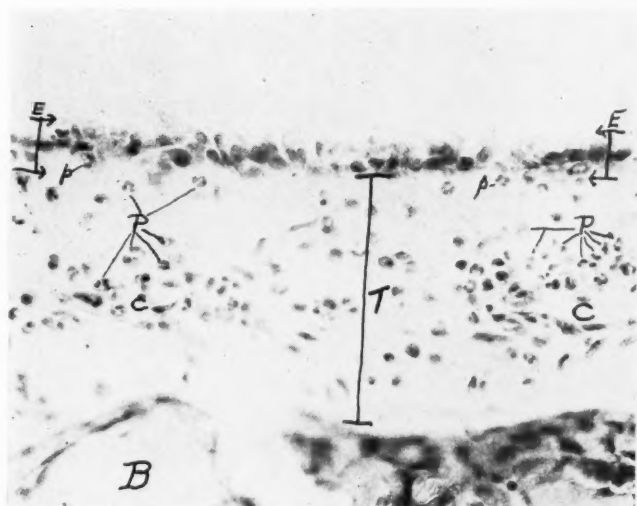


Fig. 5. Mucosa from frontal sinus of dog after contact with 10 per cent Neosilvol solution for 20 minutes, no ionization or diathermy used. Note pronounced invasion of edematous subepithelial tissue (T) by polymorphonuclear leucocytes (P) which are more concentrated around capillaries (C). (p) Polymorphonuclear leucocytes beginning to go through epithelium (E), which shows less disintegration than in Fig. 4. B—Bone.

whether this may mean that ionization is ineffectual or that normal tissues possess an ionization limit beyond which they cannot be stimulated, we are not prepared to say. Judging from the leucocytic reaction, it would seem more logical to attribute a bactericidal action, not only to the nascent ions which may be set free, but also to the subsequent phagocytosis of the bacteria by the polymorphonuclear leucocytes, which are attracted to the areas by the ions.

The marked hyperemia alone did not necessarily cause an excess of leucocytosis in the tissues.

The fact that negatively charged carbon particles suspended in a suitable electrolyte were driven into the epithelium demonstrated the power which ionization does possess to facilitate the invasion of otherwise inert foreign particles.

That certain destructive changes may be brought about was shown by the marked fragmentation of the epithelium, the edema of the subepithelial tissues and the subepithelial hemorrhages which occurred in the mucosa of the sinuses ionized with zinc sulphate solution.

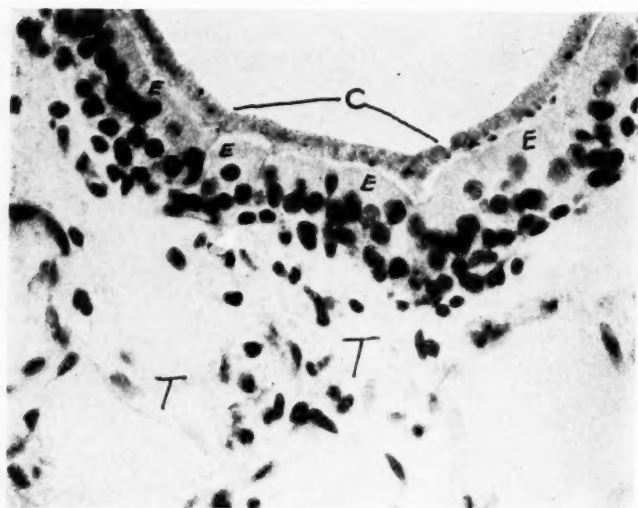


Fig. 6. Mucosa of inferior turbinate of rabbit. Carbon in suspension (C) in contact with ciliated columnar epithelium (E) for six hours, no ionization or diathermy used. No penetration into epithelium or subepithelial tissue (T).

On account of the small number of subjects studied, these results may be interpreted only as an indication of the effects which ionization of certain solutions may have upon a sinus mucosa, and will need further confirmation by more extensive experiments before definite conclusions may be drawn.

#### CONCLUSIONS.

1. Definite destructive changes were brought about in the mucosa of the frontal sinuses of dogs by the ionization of the zinc sulphate solution. These changes consisted of ballooning, fragmentation and complete destruction of the surface epithelium, a marked edema of the subepithelial tissues and an extravasation of free red blood cells into these tissues from greatly dilated and ruptured capillaries.
2. The specific response of these tissues to the ionization of silver colloids is a polymorphonuclear leucocytosis.
3. The general response of the mucosa of the frontal sinuses of dogs to a galvanic current is an engorgement of the capillaries of the subepithelial tissues.

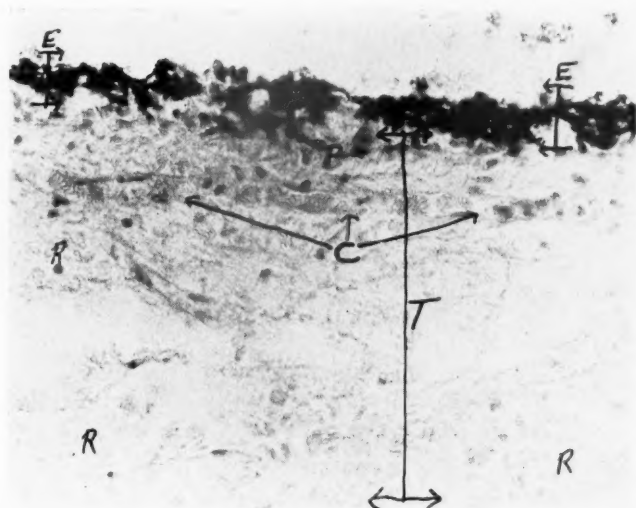


Fig. 7. Mucosa from frontal sinus of dog after contact with an India ink suspension ionized at 5 milliamperes for 20 minutes. Note penetration of epithelium by carbon particles, some of which (P) have invaded the subepithelial tissue (T). E—Epithelium showing marked fragmentation, individual cells indistinguishable due to phagocytosed carbon particles. Edematous subepithelial tissue (T) contains engorged capillaries (C) and large numbers of free red blood cells (R).

I am indebted to Dr. C. C. Bunch, Professor of Physics of Otolaryngology, and Dr. Frank H. Ewerhardt, Assistant Professor of Physical Therapy, Washington University School of Medicine, for their valuable technical advice in the conduct of these experiments.

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SYMPOSIUM ON MENINGITIS SECONDARY TO OTITIC  
OR SINUS INFECTION.\*

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LII.

A COMPREHENSIVE STUDY OF MENINGITIS SECONDARY  
TO OTITIC OR SINUS INFECTION.†

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We shall analyze a group of 623 cases of meningitis other than meningococcic or tuberculous, stressing particularly the clinical aspects. No attempt will be made to draw definite conclusions in regard to the question of vital importance, namely, the value of the different methods of treatment or prevention of meningitis secondary to ear or sinus infection.

Table I presents a complete list of our cases of meningitis other than meningococcic or tuberculous. This list may be of general interest, although infections of the ear and sinuses are not conspicuous as etiologic factors in some of the rarer forms of meningitis. In nine instances among the mixed infections there was a history of a probable primary focus of infection. These are not of sufficient importance to be given in detail.

In Table II are listed the conditions regarded as primary or as possible predisposing factors in the more common forms of purulent meningitis. This list is by no means complete, as a certain number of cases, particularly of sinusitis and mastoiditis, were undoubtedly undiagnosed. It is obvious that meningitis secondary to otitis media, mastoiditis or sinusitis, is most often due to the streptococcus. On the other hand, purulent meningitis secondary to respiratory infections is in most instances due to the pneumococcus and the influenza bacillus.

\*From the Scientific Program of the American Otolological Society, Atlantic City, April 7, 1934.

†From the Bureau of Laboratories, Department of Health, New York City.



TABLE I.

## MENINGITIS—ETIOLOGY.

Pneumococcus .....	214
Streptococcus .....	205
B. Influenza .....	118
Staphylococcus .....	30
Streptothrix .....	8
B. Coli .....	6
B. Friedlander .....	4
B. Pyocyaneus .....	4
Torula .....	3
M. Catarrhalis .....	2
Typhoid—Dysentery .....	2
Sporotrichosis .....	1
Mixed infections .....	26
Total.....	623

TABLE II.

## MENINGITIS—PRIMARY FACTORS.

	Streptococcus	Pneumococcus	B. Influenza	Staphylococcus	Streptothrix	B. Coli	B. Friedlander	B. Pyocyaneus	Torula	Catarrhalis	Typhoid— Dysentery	Sporotrichosis
Otitis media, mastoiditis and sinusitis.....	121	75	20	7	3	2	2	.....	1	.....	.....	.....
Pneumonia before onset.....	1	27	15	4	1	.....	.....	.....	.....	.....	.....	.....
Upper respiratory infections.....	8	12	4	.....	1	.....	.....	1	.....	.....	.....	.....
Pneumonia during meningitis.....	3	17	11	.....	.....	.....	.....	.....	1	.....	.....	.....
Injury to head and skull.....	8	8	4	1	.....	.....	.....	.....	.....	.....	.....	.....
Operation on head, nose or throat.....	3	4	.....	.....	.....	.....	.....	1	.....	.....	.....	.....
Bacterial endocarditis .....	4	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Brain abscess .....	1	1	.....	2	1	.....	.....	.....	.....	.....	.....	.....
Wounds, abscesses and furunculosis.....	3	.....	.....	2	.....	.....	.....	.....	.....	.....	.....	.....
Extraction of teeth .....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....
Typhoid fever .....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	1	.....
Childhood abscesses just before onset of meningitis.....	4	2	4	.....	.....	.....	1	.....	.....	1	.....	.....
General lowered resistance.....	14	11	10	5	1	3	1	.....	.....	.....	1	.....
Trauma and operations other than on head.....	.....	5	2	5	.....	.....	.....	1	.....	.....	.....	.....
No evidence of primary infection.....	33	50	48	4	.....	1	.....	1	1	1	.....	1
No history .....	2	2	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Total.....	205	214	118	30	8	6	4	4	3	2	2	1

Injuries to the head and skull have not been a particularly common factor in our series of cases. We have listed only the more serious injuries. Many of these were fractures. The other headings are self explanatory.

Table III shows the types of pneumococcus (the typing being done by agglutination and precipitin tests) in relation to the etiologic factors. It will be noted that type III was most commonly found in meningitis secondary to otitis media, mastoiditis and sinusitis.

Type I was more commonly found with respiratory infections preceding or accompanying the meningitis.

Table IV shows the grouping of the streptococcus, determined by the reaction on red blood agar. It is obvious that, with rare exceptions, meningitis is caused by streptococci of the hemolytic group.

Table V shows the species of staphylococcus found in our cases of meningitis. The staphylococcus aureus predominates.

It should be understood that a meningitis associated with a definite focus that is apparently primary is not necessarily secondary to it. Not infrequently we see cases of meningococcic meningitis following a pneumonia or otitis media which in all probability are not due to the meningococcus. Again, a recent case of meningitis caused by the hemolytic streptococcus occurred during convalescence from a pneumococcus type II pneumonia.

The symptoms in meningitis are due to various causes. They may be regarded as representing a general reaction to an acute infection and also the specific reaction due to the involvement of the meninges and the central nervous system. It is sometimes difficult to decide exactly which element produces a given symptom, the action of toxin, the increased pressure of the spinal fluid, or the active inflammatory reaction in the meninges and nerve tissue. In the production of many of the symptoms doubtless two or more of these elements play a part.

The symptoms of the forms of meningitis secondary to infections of the ear or the sinuses conform in general to the classical descriptions of meningococcic meningitis and are too well known to warrant detailed discussion. Certain points, however, may be briefly reviewed. The onset is usually sudden, but it varies greatly in its severity and is often masked by the symptoms of the primary infection. For this reason it is often difficult and at times impossible to make an early clinical diagnosis. At this stage even the examination of the spinal fluid may not be conclusive, as it may show a varying increase in cells, absence of organisms, both by smear and culture, and a normal sugar. With these findings we cannot tell whether we are dealing with a serous meningitis or the early phase of a bacterial meningitis. The early symptoms may be somewhat mild: headache, vomiting, moderate fever and perhaps a chill, slight to moderate stiffness of the neck and positive Kernig and Brudzinski signs. Or the onset may be very severe: chill and high fever, persistent vomiting, marked stiffness of the neck with positive Kernig and Brudzinski signs, and early delirium rapidly passing into coma.

TABLE III.  
PNEUMOCOCCUS—TYPES

	I	II	III	IV	VII	IX	XVIII	XIX	XXI	Group No. IV Typed	
Otitis media, mastoiditis, sinusitis .....	6	7	35	2	2	1	...	...	...	11	64
Pneumonia and other respiratory infections .....	13	7	5	...	1	...	...	...	...	14	40
Injury to head and skull .....	1	1	...	...	...	...	1	1	...	3	7
Operations on head, nose, throat .....	1	1	...	...	...	...	...	...	...	1	3
Brain abscess .....	...	...	...	1	...	...	...	...	...	...	1
Miscellaneous conditions .....	4	1	...	3	...	...	...	...	...	2	10
No evidence of primary infection .....	10	5	6	...	...	1	...	...	1	13	36
Total .....	34	22	47	6	3	2	1	1	1	44	161

TABLE IV.  
STREPTOCOCCUS—GROUPS

	Hemolytic	Non- Hemolytic	Gamma	Total
Otitis media, mastoiditis and sinusitis .....	99	5	...	104
Pneumonia and other respiratory infections .....	5	2	...	7
Injury to head and skull .....	7	...	...	7
Operations on head, nose and throat .....	2	...	*1	3
Brain abscess .....	1	...	...	1
Bacterial endocarditis .....	1	2	...	3
Miscellaneous .....	8	2	...	10
No evidence of primary infection .....	18	3	...	21
Total .....	141	14	1	156

\*Type determined by sugar reaction.

TABLE V.  
STAPHYLOCOCCUS—SPECIES

	Aureus	Albus	Both Aureus and Albus	Total
Otitis media, mastoiditis and sinusitis .....	4	...	...	4
Pneumonia and other respiratory infections .....	2	2	...	4
Injury to head and skull .....	1	...	...	1
Brain abscess .....	2	...	...	2
Furunculosis .....	1	1	...	2
Miscellaneous .....	7	1	1	9
No evidence of primary infection .....	1	2	...	3
Total .....	18	6	1	25

As the disease progresses, convulsions may occur, especially in children under 2 years of age. Stupor is found much more frequently than delirium. In a few cases the mentality is clear, well up to the end. While the pupils show reaction to light in a fairly large number of cases, when first seen, it has been observed that this reaction is usually lost as the case progresses. The knee jerks may be increased early in the disease, but they are usually lost toward the end. In a fairly large number of cases they are unequal. The muscular hyper-tonicity is most extreme in streptothrix meningitis. Of course, in

children under a year of age the signs of meningeal irritation are often very slight and hard to obtain definitely. A hemorrhagic eruption may appear in cases of influenzal, staphylococcic and streptococcic meningitis. We have been impressed on several occasions by the very high temperature ( $107^{\circ}$  F. or over) that cases of pneumococcic and influenzal meningitis show a day or two before death. The temperature does not remain at this level, but sometimes reaches it two or three times.

The course of purulent meningitis varies greatly also. At times the progress of the disease is very rapid and the patient dies perhaps in forty-eight to seventy-two hours from the onset of the symptoms. In many instances the progress is slower and the case lasts from four or five days to two or three weeks. This is particularly true of influenzal and staphylococcic meningitis.

In several cases representing all types, marked remissions of the symptoms have occurred, so that we have entertained some hope of a favorable outcome in spite of the generally accepted belief that the prognosis is almost absolutely fatal. The temperature has approached normal, the mentality has become clear, and the spinal fluid has shown a diminution in the number of organisms and a marked tendency on their part to become intracellular. In this group of cases sixteen patients have recovered: nine after infection with the streptococcus, four with the influenza bacillus, one with the staphylococcus and one with the Friedlander's bacillus. In a case due to both the meningococcus and staphylococcus recovery occurred.

The examination of the spinal fluid is essential in making a diagnosis of meningitis. The fluid is usually under increased pressure and of varying degrees of turbidity. In the later stages the amount obtained may be small if the fluid becomes very purulent or if adhesions are formed. Occasionally in fulminating cases the fluid may be only slightly hazy or even clear, the smear showing very few cells and an overabundance of organisms. Usually the cell count varies from a few hundred to many thousands, with polymorphonuclears predominating. The chemical tests show varying increase in the albumin and globulin. The sugar may be normal early in any type of meningitis. As the disease progresses, the amount of sugar markedly decreases or may entirely disappear. The bacteriologic study of the spinal fluid is by far the most important part of the examination, and unfortunately one that is done most inadequately in many laboratories. The examination of the stained smear is often inconclusive. For an absolute diagnosis, we must obtain the organism by culture.

In certain cases of pneumococcic meningitis, however, the type can be determined directly from the spinal fluid.

Blood cultures were made in sixty-eight of our cases. Of these, thirty-two were positive. In the group associated with ear or sinus infection there were thirteen positive blood cultures out of thirty-eight cases. It is obvious that repeated blood cultures must be made in order to be certain of the absence of organisms in the blood stream. It was usually difficult to determine whether the bacteremia preceded or followed the meningeal infection. Unquestionably, the presence of bacteremia adds to the gravity of the prognosis.

It may be well to comment briefly on meningitis sympathica or serous meningitis. We use these terms interchangeably to describe a meningeal condition occasionally found when an active infection, such as an otitis media, sinusitis or brain abscess, occurs near the meninges, without actually invading them with the infecting organism. Well marked signs of meningitis are present and the spinal fluid shows changes probably due to the reaction of the meninges to the contiguous inflammatory focus. These changes consist of an increase in the amount of fluid which is usually hazy or cloudy, an increase in cells which may reach several thousand, with polymorphonuclears usually predominating, an increase in the albumin and globulin and a normal sugar content. There are no organisms by smear or culture. This condition may remain as an entity and go on to recovery or it may represent the early stage of a true bacterial meningitis. The importance of prompt and thorough eradication of the primary focus of infection is quite obvious.

The case fatality in meningitis secondary to infection of the ear or sinus is so high that serious consideration should be given to methods of prevention. It seems to us that much could be accomplished by the education of the public in regard to the serious after effect of the neglected common cold and the importance of routine examination and early treatment of infections of the nose, throat and sinuses.

We have already referred to the necessity of prompt and thorough eradication of primary foci of infection, especially when meningeal signs occur.

Reference must be made to Kolmer's recommendation of pre-operative immunization by means of serums or vaccines to prevent meningitis. It will be difficult to prove the value of these measures, since meningitis is comparatively rare after operations on the mastoid or sinuses. Most of us would hesitate to give serum for prophylaxis because of the danger of sensitizing the patients. To produce an

active immunity by the use of vaccines would require a longer time than is usually available. Moreover, Kolmer bases his theory on the results of experimental work with animals. The conditions which obtain experimentally are quite different from those in the human being.

A great variety of procedures has been resorted to in the treatment of these forms of meningitis. It would seem from our own experience and from a study of the literature that the most important therapeutic factor, aside from the removal of the primary foci of infection, is adequate drainage of the cerebrospinal fluid. Unless there is blocking, this can be accomplished by lumbar puncture. There is no evidence that the more radical forms of drainage are more effective. A greater number of recoveries has followed ordinary spinal drainage than any other form of treatment.

During the last few years the use of forced spinal drainage has been recommended. We have employed this method in twenty-three instances. In a few cases there was a brief period of improvement. In two recent instances the necropsy showed an extensive edema at the base of the brain, although there appeared to be free drainage of fluid before this method was applied. Great care, therefore, must be exercised in using this procedure in cases of meningitis, where adhesions may interfere with adequate drainage of the increased amount of spinal fluid.

The success that has followed the use of antimeningococcic serum in meningococcic meningitis has made it logical to use specific serums, when available, in the treatment of other forms of meningitis. We have used such serums in many of our cases intraspinally and also intravenously if a bacteremia was present. On the whole, the results have been discouraging. In a fairly large percentage of cases there has been a period of improvement, and in a few instances recovery has taken place. Usually we did not believe that the recovery was due to the serum. In two cases, however, in which a hemolytic streptococcus meningitis followed scarlet fever complicated by otitis media, we believe we were justified in attributing the recoveries to the specific action of the antiscarlatinal serum. We continue to use and advise the use of specific serums for whatever value they may have. It is possible that in the future, more potent serums may be developed.

We have also used autogenous vaccines, both intraspinally and subcutaneously in a small number of cases. Two recoveries, one a staphylococcic meningitis and one an influenzal meningitis—followed

the use of the method. In most instances, the course of the meningitis is too short to admit of the use of this measure.

The use of various chemical agents has also been advocated by a number of workers. We have injected intraspinaly gentian violet, acriviolet, acriflavin and ethylhydrocuprein hydrochloride (optochin), both alone and in combination with serums. It is our impression that these agents are of doubtful value and occasionally may be harmful. We have, therefore, discontinued their use with the exception of optochin in pneumococcic meningitis. There has been a tendency of late to employ the intracarotid route for the injection of serum and chemical agents in cases of meningitis. We have never used nor advocated this method. In the first place, we can see no advantage from its use, since the rapidity of the circulation of the blood is so great. In the second place, one instance has been under our observation, and others have been reported to us in which thrombosis of the cerebral vessels followed this method of injection. We therefore regard it as a dangerous procedure.

Several factors contribute to the difficulty in successfully treating this group of cases. The most important of these is the fact that the meningitis is usually secondary to one or more primary foci of infection which as a rule cannot be completely eradicated. There is therefore a constant reinfection of the meninges. The frequent presence of a bacteremia adds to the gravity of the situation. Furthermore, there is much that is unknown in regard to the bacteriology of these organisms, particularly as regards the virulence of the various bacteria, even though they belong to the same type. It may well be that recoveries are often due to the fact that the infecting organism was of relatively low virulence rather than to the treatment that was applied. Then, too, the unpredictable resistance of the host must be considered. In other words, the pathogenicity of bacteria does not depend so much on their own inherent biologic characteristics as it does on the complicated reaction of host and organism. This cannot be overemphasized.

No specific procedure can therefore be formulated for the treatment of these forms of meningitis. Early and repeated spinal drainage may be regarded as an important factor in the management of these cases. When specific serums are available, it seems logical to employ them intraspinaly. Surgical intervention should, of course, be resorted to for the removal of definite suppurative foci. Other therapeutic measures must, in the light of our present limited knowledge, be regarded as experimental. Finally, one must always bear in mind the possibility of spontaneous recovery.



## SUMMARY.

1. An analysis has been presented of 623 cases of meningitis other than meningococcic or tuberculous.
2. Tables have been given showing the relation of these cases to probable primary sources of infections.
3. The relative incidence of various organisms in this group of cases has been tabulated.
4. The clinical features and the laboratory diagnosis have been discussed. Special mention has been made of serous meningitis. We have referred to sixteen recoveries in this group of cases.
5. The problems of prophylaxis and treatment have been considered, outlining the methods of treatment that we have employed, without drawing definite conclusions.

### LIII.

## BACTERIOLOGY OF MENINGITIS FOLLOWING OTITIS MEDIA AND RELATED INFECTIONS.\*

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The relative incidence of microbes found in the spinal fluids from cases of meningitis studied by us, other than those due to meningococcic or tubercle bacilli, is given in Table I of Dr. Neal's paper.

This table shows that our findings agree with those of bacteriologists in other parts of the world, in that the great majority of such meningitis cases occur after middle ear and sinus infections and that the vast majority of these are due to the streptococcus, pneumococcus or influenza bacillus.

We have studied about 600 cases showing each a single kind of organism, and during this time have studied in addition over a thousand cases of meningococcic meningitis and about a thousand cases of tuberculous meningitis, as well as a number of cases of mixed infections, showing the relative frequency of these cases.

Of the 597 cases reported in this paper, 205 were due to streptococci, 214 to pneumococci and 118 to influenza bacilli. The remainder, 60, fall into nine groups each due to a different kind of micro-organism.

For the identification of these microbes we use stained spreads, cultures, serologic reactions and animal injections.

We may determine by stained spread of the centrifuged spinal fluid that the organisms seen under the microscope belong probably to one of the large general groups of microbes. We cannot, however, always be sure of this, since bacteria and their relatives often present an atypical picture in stained spreads. For instance, actinomyces may look like streptococci, streptococci may look like bacilli or like pneumococci. There is no such organism as streptococcus mucosus capsulatus; on culture and on serologic test an organism so called proves

\*Presented as part of Symposium on Meningitis Secondary to Otitic or Sinus Infection at the annual meeting of the American Otological Society, Atlantic City, April 7, 1934.

usually to be pneumococcus type III. Therefore, in addition to making an examination of the direct spread, cultures should always be made and studied.

In the case of the less frequent kinds of organisms found it is sufficient from a practical standpoint to determine only the genus, for the laboratory can at present give no further help to the clinician. But the case is different with the first three groups found—streptococci, pneumococci and influenza bacilli.

Here the question of determining the agglutinative types of the strains found is practically an important one. For the agglutinative type is a guide to the type of antibacterial serum to use. The question of making this test quickly, easily and trustworthily is of the greatest importance. This has occupied the attention of laboratory workers for many years. As regards the pneumococcus we have the test on a very satisfactory basis. We know the numbers and distribution of many types. We know there is no longer a group IV, but that among this group there are a number of types some of which have been shown by Miss Cooper and her associates to be the cause of cases having a significant mortality and for which antisera have been manufactured and used with encouraging results. Thus, a case of meningitis following pneumonia was found by Miss Cooper to be due to her pneumococcus type XII. The patient was given Cooper type XII serum and recovered.

Recently a quick method of determining types of pneumococci has been developed based upon the "Schwellings phenomenon," mentioned so long ago (1902) by Neufeld. This has proved to be a rapid and trustworthy method of demonstrating agglutinative types of pneumococci in material directly from the patient—in sputum, pus, spinal fluid and so on.

We have made some advances, too, in our knowledge concerning the kinds of influenza bacilli causing meningitis. Dr. Povitzky, working in our laboratory during the great epidemic, found that among the strains of influenza bacilli causing meningitis an agglutinative type grouping occurred. And Dr. Neal and her associates have demonstrated that the antiserum developed from this type gives evidence of having some curative effect in cases of meningitis due to this type.

Streptococcus typing is not on so satisfactory a basis as is pneumococcus typing. This is indicated by Dr. Neal's table, showing the grouping of these bacteria by appearance on blood agar plates only. We know little about the incidence of agglutinative types among

hemolytic streptococci, particularly in these cases. And until we do know we cannot decide how worth while it is to prepare antibacterial serums of a particular type. Some investigators have reported finding a type group among meningitis strains. They have even given such a group the name streptococcic meningitides. We have not been able fully to corroborate such a specific grouping. We studied thirty-seven strains of streptococci from cases of meningitis isolated by Neal and Gosling, and found that eleven of them fall into our largest scarlet fever group (Type Sub I), another into our scarlet fever Type IV, two into our erysipelas Type I group, and a small group of seven forms a new agglutinative group. If members of this agglutinative group occur frequently enough in streptococcus infections an antiserum should be manufactured for use in these cases. It is significant that so many cases were due to bacteria strains that fall into our large scarlet fever group, and that among these cases Dr. Neal had two cures following the use of our antiscarlet fever serum. We would expect many of the strains from cases of this type to be related to the scarlet fever strains, since middle ear infection is so frequent a secondary infection in scarlet fever.

Of course the question of the treatment of these streptococcus cases is complicated by several factors relating to the nature of these cocci and to the time at which the cases were usually seen. Pathogenic streptococci produce two types of poisons which have been called respectively exotoxins and endotoxins. The exotoxins stimulate the production of antitoxins, effective in scarlet fever. The endotoxins stimulate the production of antibacterial substances effective against the invasive forms of these cocci. So in order to be effective in meningitis which is due to invasion of the organism a serum must contain potent antibodies against this second type of poison. Practically no investigators other than ourselves have stressed this point, and no standard for potency of this type serum has been worked out as it has for the pneumococcus serum.

Our scarlet fever antitoxin serum is manufactured by a method that stimulates the production of both types of antibodies, but horses respond differently to these two antigens, and since we have no standard method of determining the potency of antibacterial serum we are never sure that we have chosen a good horse for producing equally well the two types of antibodies. The anti-exotoxic part of the serum probably has little effect on invasive streptococcic infections. It is the antibacterial serum—that is, the anti-endotoxic part, from which we hope for the best results in these cases—and such a serum must be type-specific.

We know how to manufacture such a type serum. We simply lack funds for applying that knowledge.

Then the question of seeing the cases early enough has to be considered. We practically never know of cases early enough to hope for good results from serum treatment.

This brings us to the question of bacteriologic work in prevention. It would seem as if an ear infection might be an ideal condition to attempt to immunize against further invasion. But here again nothing has been done to determine the type of infection present in otitis and sinusitis. Of course we can use an autogenous vaccine but this takes time to prepare.

Dr. Kolmer in his extensive work on the vaccination of such patients does not mention the importance of knowing the agglutinative type of coccus present and the use of that type for the vaccine. He has investigated the advisability of using preliminary injections of autogenous vaccines as a pre-operative preparation to raise the immunity to a point where some protection may be afforded against meningitis. He claims that this is possible.

In conclusion I may say that in order to test the worth of preventive and curative measures in treating invasive forms of microorganisms in otitis media and related infections we must know more about the agglutinative types of the organisms producing such infections.

#### SUMMARY.

Of the 597 cases of meningitis other than those due to meningococci or tubercle bacilli, 205 were due to streptococci, 214 to pneumococci and 118 to influenza bacilli. The great majority of these cases occur after middle ear and sinus infections.

The importance of determining the serologic types of these microbes is emphasized. In the case of the pneumococcus the identification of type is on a satisfactory basis. With the influenza bacillus some advance has been made. But we know little about the incidence, either seasonal or geographic, of agglutinative types among hemolytic streptococci, particularly in these cases. And until we do know we cannot decide how important it is to prepare antibacterial serums of a particular type.

The authors studied thirty-seven strains of streptococci from cases of meningitis for agglutinative types and found that twelve of them fall into their scarlet fever serologic groups, two into their

erysipelas Type 1 group and a small group of seven forms a new agglutinative group.

The authors conclude that in order to test the worth of preventive and curative measures in treating invasive forms of micro-organisms in otitis media and related infections we must know more about the agglutinative types of the organisms producing such infections.

PATHWAYS OF INFECTION IN SUPPURATIVE  
MENINGITIS.\*

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The opinions and data I wish to present are those of a general pathologist who has, for fifteen years, had the privilege of being associated with an eye, ear, nose and throat hospital, which has offered a wonderful opportunity for the observation and study of one of the most tragic diseases in medicine. These associations have resulted in the formation of conclusions which may be worthy of review before a Society frequently confronted with the complication of suppurative meningitis.

What I have to say, and more, might easily be found if reference is made to the previous published works of MacEwen, Fraser, Eagle-ton, Friesner, Maxwell, Kopetzky, Turner and Reynolds, and others. It is difficult for one to write upon this subject without being influenced by the convincing data and conclusions drawn by these authors, and it would appear that what I have to say is highly redundant; however, my attempt is to add additional material and conclusions to the facts already established by those authors who have already devoted so much effort and special study to this subject.

A review of the intracranial suppurative diseases for the past eight years at the Manhattan Eye, Ear, Nose and Throat Hospital has been made. This has been accomplished chiefly by an analysis of the deaths which have occurred during the period of 1926-1933, inclusive. In this interval there were 363 deaths in the institution. Of these there were 110 cases of suppurative meningitis, 53 cases of brain abscesses and 14 cases of both brain abscess and meningitis. Thus, there were a total of 177 cases of suppurative intracranial disease, or 48.8 per cent of all the deaths in the institution. Of these, 107 were males and 70 females. The majority of the cases occurred between the ages of 1 and 10. The detailed data of these cases are presented in the following table.

\*Presented as part of Symposium on Meningitis Secondary to Otitic or Sinus Infection at the annual meeting of the American Otological Society, Atlantic City, April 7, 1934.



Total deaths for eight-year period between 1926-1934: 363.

DEATHS FROM INTRACRANIAL SUPPURATIVE DISEASE.

1. Meningitis	110
2. Meningitis and Brain Abscess	53
3. Brain Abscess	14
Total	177 or 48.8%

PATHWAYS OF SUPPURATIVE MENINGITIS.

1. Auditory Apparatus	90
2. Paranasal Sinuses	18
3. Cavernous Sinuses	8
4. Postoperative	8
5. Miscellaneous	3
Total	124 or 70%

PATHWAYS OF BRAIN ABSCESS.

1. Auditory Apparatus	37
2. Paranasal Sinuses	18
3. Osteomyelitis, etc.	3
4. Miscellaneous	9
Total	67 or 30%

DISTRIBUTION AS TO AGE AND SEX.

Age	Cases	Male	Female
1-10	42	107	70
10-20	33		
20-30	31		
30-40	20		
40-50	28		
50-60	18		
60-70	5		

Aside from the cases of deaths, there were eight cases with clinical symptoms of meningitis which recovered, but these failed, pathologically, to meet all the diagnostic requirements of an active suppurative process. There were ten cases of brain abscess which recovered.

In the remarks which follow, suppurative meningitis and brain abscess will be discussed in conjunction, as in these conditions the infection enters through similar pathways.

In compiling these data a study was also made of the intracranial suppurative disease in relation to nasal infections, and a brief resumé may be appropriately included in this presentation.

In reviewing these cases, the following structures were concerned in the spread of the infection from the regional soft tissue and bony structures to the intracranial organs.

The regional venous circulation is of prime importance in the spread of infections to the intracranial structures. This takes place in most instances by means of a septic thrombosis. The process is one of progressive damage to the endothelium of the venous channels with a clotting of blood and septic thrombosis. This may occur against the circulation, of a retrograde nature.

Inflammatory processes in the bones of the skull, paranasal sinuses and particularly of the auditory apparatus are usually the foci from which the infection spreads by way of the venous circulation or through the contiguous tissues to the leptomeninges and brain.

The lymphatic circulation is of questionable significance as a pathway of infection from the peripheral parts to the intradural structures, because lymphatics connecting these structures have not been satisfactorily demonstrated.

There are certain natural pathways which provide avenues for the passage of micro-organisms to the leptomeninges. Of particular interest in this connection is the direct continuity of perineural spaces of the auditory, vestibular and olfactory nerves with the pia-arachnoid membrane.

There may be direct extension of the infection to the meninges through a fracture of the skull by accident or operative trauma.

Furthermore, intracranial infections occur from distant organs by means of bacterial emboli. One case in our series developed a cerebral abscess secondary to a chronic bronchitis and bronchiectasis. A metastatic pneumococcal meningitis is not an uncommon complication of lobar pneumonia.

These pathways will now receive further consideration as related to primary infections of the orbital, nasal and auditory structures. The venous circulation in the region of the orbit and associated nasal sinuses has received the most thorough investigation, because of its relation to the cavernous sinus. The spectacular course and serious nature of cavernous sinus infection is well recognized.

In our series there were eight cases of cavernous sinus thrombosis, as follows: Two followed infections of the nose; one of the cheek; two of orbital abscess and cellulitis; one was secondary to sphenoid and ethmoid sinusitis. Two cases of cavernous sinusitis were of otitic origin, as a result of retrograde extension through the petro-

sal veins to the cavernous sinus. These are of great interest, because the petrosal veins are efferent vessels of the cavernous sinus and the infection traveled against the circulation.

Following a cavernous sinusitis the infection finds a ready pathway to the leptomeninges through other tributary veins connected with the leptomeninges. When the venous structures of the brain become involved a septic phlebitis results and usually the walls of the veins rupture, allowing a diffuse distribution of the infection to the meninges. With acknowledgment to Turner and Reynolds, I present their instructive demonstration of the venous blood supply associated with the cavernous sinus.

There were thirty-one cases in our series of intracranial complication following nasal sinusitis, eighteen of which died of meningitis, eight of frontal lobe abscess. Five cases of abscess recovered after proper treatment. Of the eighteen cases of meningitis, six were post-operative. Autopsy findings in many of these cases revealed bony dehiscence leading through the dura and into the pia-arachnoid area. Whether the opening was traumatic or the result of a pre-existing low grade osteomyelitis of the bone is frequently very difficult to determine. If there is extensive disease in the paranasal sinus with a low grade osteomyelitis, the bone is osteoporotic, and is easily fractured by the gentlest manipulation. From these cases, it appears that many postoperative meningitic cases following nasal sinusitis result from a pre-existing disease of the bone.

Suppuration or trauma to the frontal and ethmoid sinuses are most commonly followed by an osteomyelitis or a meningitis. However, the sphenoid and maxillary sinuses are occasionally the nidus of infection. When intracranial complication occurs as a secondary process to infections of the paranasal sinuses the pathway of infection is usually by contiguity of tissue or by means of a suppurative phlebitis. While it is true that infection of the bones of the nose is a frequent source of intracranial disease, it is equally true that osteomyelitis of the calvarium with massive areas of necrotic bone rarely produces a suppurative meningitis. This is probably due to the peculiar distribution of the venous channels of the dura and diploic structures of the calvarium. In the case of chronic suppuration of the frontal air cavities there may arise cariesnecrosis of the bony walls with a sequestration of bone resulting in a defect in the posterior wall, followed by an epidural or frontal lobe abscess or meningitis. Intranasal trauma involving the sheaths of the olfactory nerve filaments may easily be followed by infection of the perineural

sheaths and the passage of the infection along the sheath to the pia-arachnoid membrane through the cribriform plate. The perineural sheath of the olfactory nerves has not been the avenue of infection in any of our series; however, such cases have been reported by Turner and Reynolds. In fact, it has been fairly definitely proven that the infective agents of infantile paralysis and the meningococcus in epidemic meningitis pass through the olfactory perineural sheath.

Infection associated with the auditory apparatus has always been known to be the chief source of suppurative intracranial disease. Of 177 deaths from suppurative disease in our institution, ninety cases of meningitis were secondary to infection of the auditory apparatus, and thirty-seven cases of brain abscess were secondary to a primary focus in the temporal bone. The pathways of infection in these were not easily determined; clinically and pathologically labyrinthitis and petrositis were the chief inflammatory processes subsequently complicated by intracranial suppurative disease.

The pathways of infection from disease of the temporal bone are numerous and complicated and offer a fruitful field for research in an attempt to explain the sequence of events as the infection spreads from the tympanic cavity of the ear to the intracranial structures.

The venous supply of the auditory apparatus has received detailed study by MacEwen, Siebenmann, Pietrantonio, and of additional interest is the recent publication by Ruskin. The venous supply of the temporal bone is of particular interest in connection with the spread of infection from the mastoid auditory cleft and petrous bone to the associated structures of the skull. For years the lateral sinus has been of tremendous interest to the otologist, as many infections of the mastoid and ear result in septic sinus thrombosis and may spread through the wall and contiguous tissues to the meningeal structures, resulting in either meningitis or brain abscess.

The infection may also spread along tributary veins of the lateral sinus to the cortex of the brain or cerebellum and not infrequently most of the venous channels of the dura may become involved in a suppurative process. In this connection, I recall a case of mastoiditis with septic sinus thrombosis which was properly treated surgically, but disastrous events followed one after another in the following sequence: A homolateral sinus thrombosis, followed in the course of a few days by cavernous sinus thrombosis of the contralateral side revealed by all the usual symptoms of these complications. Death came to the patient in a few days with the classical symptoms of meningitis. Autopsy confirmed the clinical diagnosis,

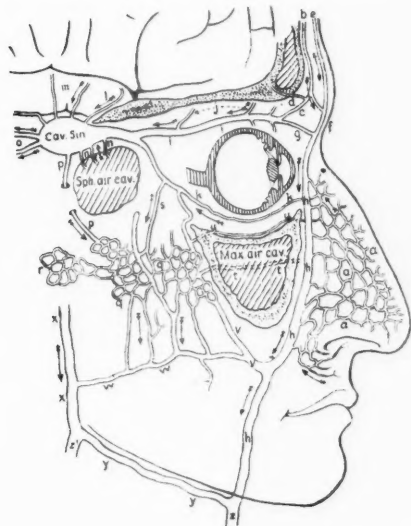


Fig. 1. Diagrammatic representation of the afferent and efferent venous channels of the cavernous blood sinus. After Turner and Reynolds.

revealing a suppurative infection of the lateral sinuses of the operative side with septic thrombosis of the inferior petrosal vein of the same side, extending into both sides of the cavernous sinus. There was pus in the opposite petrosal vein and yellowish purulent suppuration of the opposite lateral sinus and a diffuse suppurative meningitis.

The venous channels associated with the middle and internal ear and the petrosal portion of the temporal bone are numerous and of very complex structure and anastomosis, thus suppurative infection may spread along the veins of the lateral sinus, inferior and superior petrosal veins, cavernous sinus, carotid and pterygoid plexuses of veins, and then indirectly to the superficial veins of the entire dura and nervous system. The venous connection of the labyrinthine and tympanic cavity and petrous pyramid are the chief factors in the spread of infection of the mastoid and middle ear to the adjacent intracranial structures, resulting in a septic phlebitis of the superficial cerebral vessels and followed by acute purulent leptomeningitis. However, at times, a very extensive involvement of the veins and sinuses of the dura may occur, and unless there is a rupture of the suppurative thrombi into the pia-archnoid meningitis or abscess may not supervene.

## REPORT OF A CASE.

July 15. The patient, J. S., first consulted Dr. Moghtader, complaining of pain in the left ear which he had had for two days. No temperature. The ear drum was bulging and slightly red. Myringotomy was performed at the hospital.

July 21. Second myringotomy was performed. There was no rise in temperature. The patient was discharged from the hospital after ten days.

August 11. Patient again admitted to the hospital. Mastoidectomy was performed with no exposure of the sinus or dura. The deeper cells were quite diseased.

August 31. The patient complained of headaches the past few days. Temperature was normal and mastoid wound looked clean. More pain the next three days.

September 3. Temperature up to  $101.2^{\circ}$  for the first time. The same day the temperature gradually went up to  $104.4^{\circ}$ , with pulse 130 and respiration, 28. At night the patient complained of more pain and was drowsy. Refused to eat. Perspired very much and vomited. Next day the mastoid wound was opened. Both the sinus and dura were exposed. Lumbar puncture performed with normal laboratory report. Patient felt better for the next three days.

September 9. Patient complained of feeling chilly, but had no actual chill. Complained also of rigidity of neck and more pain over the right eye. Temperature went up to  $102^{\circ}$ . The lateral sinus was opened, a clot was removed, free bleeding established and the jugular vein tied off. Culture of the clot showed no growth. During this operation the dura was nicked at the knee and cerebrospinal fluid escaped. Seven hundred cc. of saline solution given intravenously, and Murphy drips of 5 per cent glucose. Temperature came down to  $100.4^{\circ}$ . Three days later the temperature went up to  $102.6^{\circ}$ . Patient had more pain and became irrational. The following day paralysis of the external rectus and drooping of the eyelids was noticed. There was a slight rigidity of the neck with positive Kernig. Temperature,  $102.8^{\circ}$ . The following day, cerebrospinal fluid was drained at the point where the dura had been nicked. It drained for 24 hours. Specimen sent to the laboratory. Culture showed no growth. For the next six days the temperature kept up between 101 and 103 degrees, with pain in back of the head and neck. Blood cultures taken were negative.

September 23. The patient was operated on again. This time more bone was removed and more of the dura exposed. The lateral sinus was split open almost as far back as the torcula. An intravenous injection of 700 cc. saline solution was given.

September 27. The left eyelid was swollen and in 48 hours the eye was protruding and fixed with the vision down to less than 5/200. The left side of the face and neck was swollen. Thinking that this might be an orbital abscess, an incision was made over the lid and the socket investigated. No pus was found. The temperature was still of a septic character, with pulse between 108 and 120, and the respiration around 24. The patient showed some symptoms of absorption. He acted queerly and had involuntary micturition. His condition improved slightly from October 3 to 8.

October 9. The patient complained of severe pain on the right side of the chest. Dr. Stowell thought it might be an infarct. The next few days the patient coughed and expectorated a lot.

October 16. A great deal of pus was expectorated. X-rays taken showed the presence of an abscess in the right lower lobe. This condition was left alone to

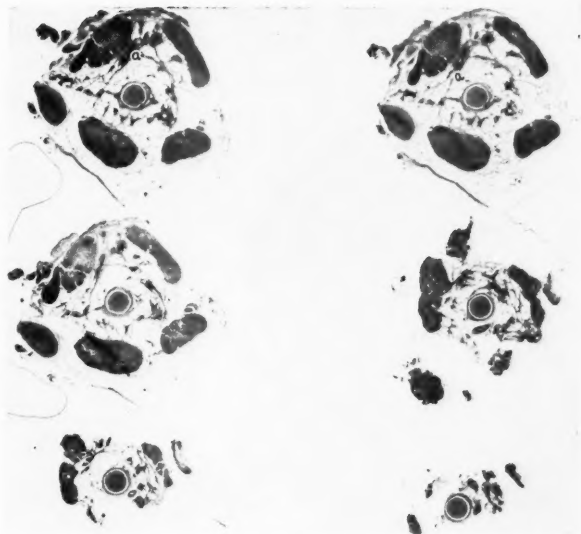


Fig. 2. Several serial sections of retro-ocular tissues in a case of cavernous sinus thrombosis of a child 8 years old, with a furuncle of the upper eyelid. The thrombosed ophthalmic vein is shown at (a) and an abscess at (b).

be taken care of by the patient himself. During the next few days, there was an improvement in the condition of the eye. The swelling had gone down considerably and the eye was not fixed as much as before. There was some swelling on both sides of the neck.

October 28. The swelling on the left side of the neck extended to the face right up to the lower lids. The skin was glossy and very hard. The general condition of the patient was very poor at the time. He perspired freely; had an irregular pulse; became exhausted very easily. Then he began to complain of soreness in the throat and difficulty in swallowing. Gradually, in about three or four days, the swelling on the left side of the face went down. A retropharyngeal abscess was opened, which later on was found to be communicating with the abscess cavity at the site of the jugular incision. Culture of the pus showed very scanty growth of short-chained nonhemolytic streptococci.

November 8. The patient developed a pneumothorax.

November 20. A rib resection for empyema was performed. The next day the patient was transfused, 600 cc. The swelling of the left eye, which had gone down considerably, showed signs of recurrence, but this time the swelling and chemosis were confined to the lower conjunctiva.

November 21. This was incised and a great deal of pus removed (from the orbit). The next few days the patient seemed to improve some as far as the general condition was concerned.

November 29. The swelling on the left side of the neck began to increase. The patient complained of headache over the left eye and over the left ear. Had





Fig. 3. Microscopic section of the left retro-ocular tissues of J. R. with multiple furuncles on the nose, and bilateral cavernous sinus thrombosis. There are numerous abscesses present in the muscle and fatty tissues as indicated (a). Septic thrombosis of ophthalmic vein is shown at (B).

difficulty in opening the mouth. In other words, the same condition was developing on the left side as on the right.

December 4. This abscess was opened from the inside by an incision just in front of the tonsil. There was profuse discharge. An X-ray of this abscess cavity was made after injecting lipiodol. It showed the presence of two different cavities communicating at the upper end (horse shoe). This was treated by pressure from outside and suction from within. A 2 per cent solution of mercurochrome was instilled in the cavity at each treatment. During the next three or four weeks the condition of the patient showed improvement. The left side of the neck, which had been swollen, appeared almost normal. There was very little discharge from the abscess in the throat. The eye was very much better. The mastoid wound looked clean and there were no new foci of infection. The patient began to put on weight. His appetite improved. The small hernia of the brain that had come on began to get larger. During this period the patient had two transfusions, one on December 16 and one on January 3, about 500 cc. each time.

January 8. Patient sat in chair for about ten minutes for the first time, and thereafter daily.

January 19. Conditions changed again and the patient showed signs of absorption; slight elevation of temperature. He complained of dizziness and headaches at times. The swelling on both sides of the neck recurred. He was operated on and the abscesses on both sides of the neck were opened.

January 23. The patient complained of earache on the left side. He thought he couldn't hear well with that ear. Examination showed no pathology.



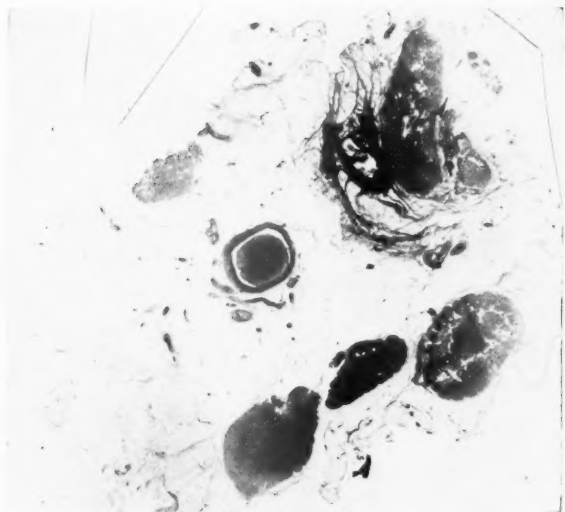


Fig. 4. Sections of same case, J. R., of the right retro-ocular tissues, showing marked septic thrombosis of the ophthalmic vein. The optic nerve on this side is not as swollen as that of the opposite side, and the protosis while definite was not as marked as on the left side.

February 1. Had a chill that lasted five minutes. The temperature went up to  $102^{\circ}$ . For the next five days he felt all right. Then he began to complain of frontal headaches which increased later on. He wasn't feeling as strong as he had been and did not want to sit in the chair. He vomited at times; perspired freely, mostly over the extremities.

February 19. A lumbar puncture was made for diagnostic purposes and also for removal of pressure. The fluid was under terrific pressure. About three ounces must have escaped. Soon after, the patient complained of severe headache. In twenty minutes he was in a state of coma with typical Jacksonian epilepsy. Twitching and convulsions started at the left side of face and spread to the left side of the body. These attacks came on repeatedly at very short intervals. These attacks were subdued with chloroform and sodium amytol injections. Temperature gradually went up to  $105^{\circ}$ , pulse to 160, and respiration to 46. The patient died in twenty hours without regaining consciousness.

The fundi were examined several times during the patient's illness. The last examination was done on February 13. It showed papilledema of about four diopters in the left eye, two in the right, with atrophy; vessels engorged. Macula and the periphery were normal. In the earlier examinations there were some exudates which cleared up later. The fields taken showed definitely evidence of a progressive atrophy. The blind spots were enlarged.

#### AUTOPSY REPORT.

The body is that of a white man, 37 years of age, well developed and well nourished. There is a slight swelling and drooping of the left eyelid. There is a

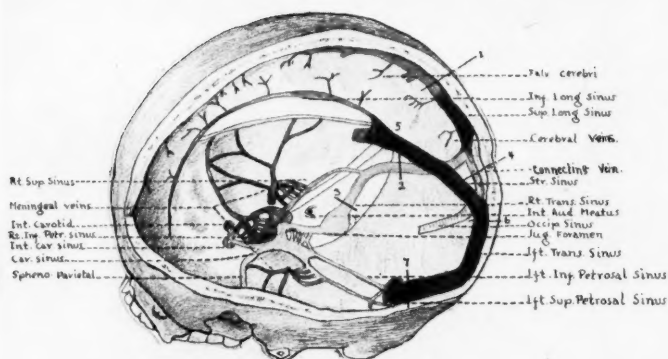


Fig. 5. Schematic representation of multiple sinus thrombosis in the case of J. S. There is no sinuum confluens, and according to Dr. J. R. Page, it is usually not present. The numbers represent the areas, sectioned microscopically.

brain hernia 3 cm. in diameter over the right mastoid region. There are numerous operative wounds in the region of the right mastoid which are partly healed. There is a recent operative wound in this region which is packed with gauze. There are several operative scars over the right jugular and right side of the neck. There are several operative wounds on the left side of the neck. There is an operative wound of the right chest posteriorly where there has been an empyema drainage.

On removing the skull there is considerable diploic congestion of the occipital bone. The dura is adherent to the skull in the region of the mastoid bone and lateral sinus operation and around the brain hernia. Upon removing the dura and cortex, a small amount of clear spinal fluid escapes. There is some congestion of the pial blood vessels, but no distinct inflammatory exudate is noted, either on the cortex or at the base of the brain. There is considerable congestion around the base of the brain.

Upon removing the brain, the dura, in the right middle fossa is diffusely yellowish brown in color. In the left, there is some congestion in the dura over the sphenoid bone and in the region of the cavernous sinus. This appears to be an old inflammatory hemorrhagic condition. In the posterior pole of the right occipital lobe of the brain there is a hemorrhagic necrotic area of brain tissue, 3 cm. in diameter, from which area there was a cerebral hernia which projected into the mastoid wound and was adherent and covered with skin epithelium. The superior longitudinal sinus is patent up to the region of the pachionian bodies. In this region there is a dark red clot in the sinus which is slightly adherent, and the dura in this region is hemorrhagic and copper colored over an area 10 x 5 cm.

The superior longitudinal sinus continues backward and to the right of the midline, passing diagonally across the posterior fossa of the skull and the right occipital bone to form the right lateral sinus. It passes out 2 cm. to the right of the internal occipital protuberance, where it bends to form the right transverse sinus. At this angle there is a connecting vein to the angle of the left lateral sinus.

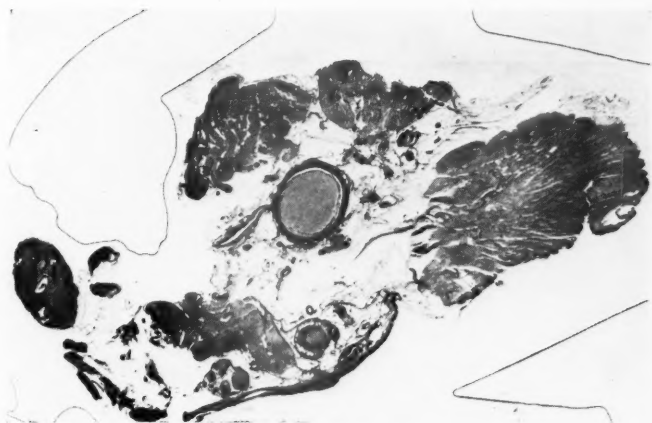


Fig. 6. Section of left retro-ocular tissues in the case of J. S., showing at (a) thrombosed and organized ophthalmic vein, with disappearance of the acute symptoms.

This connecting vein apparently takes the place of the torcular or sinum confluens. The posterior portion of the superior longitudinal sinus is filled with a dark red blood clot. In the region where the connecting vein is given off the sinus is fibrotic and the connecting vein is likewise practically entirely obliterated by a thrombophlebitis. The right transverse sinus is obliterated with fibrous tissue, as is also the right lateral sinus which was operated upon and obliterated. This portion of the sinus remains as a fibrous cord. The right inferior petrosal sinus is likewise thrombosed, as is the external third of the superior petrosal sinus. There appears to be an obliteration of the veins connecting the inferior petrosal sinuses upon the upper basi-occipital surface. The right cavernous sinus appears patent but the left is fibrotic and partially obliterated. This is the side where there was proptosis of the eye and other symptoms associated with cavernous sinus thrombosis. The left inferior petrosal contains a septic thrombus consisting of fibrin and pus. The purulent exudate is more marked in the external portion of the sinus where it connects with the left lateral sinus. The left lateral bulb and sinus are completely filled with free pus. This extends to the left transverse sinus and to the terminal portion of the straight sinus of which the left lateral sinus is a continuation. The septic thrombus involves, at this point, the connecting vein to the right transverse sinus. The posterior two-thirds of the straight sinus shows an organizing suppurating thrombus, while the anterior one-third shows an adherent dark red thrombus. There are no signs of active meningitis.

The cerebellum shows some engorgement of the veins; the inferior surface shows two circular grooves resulting from pressure of the tuberosities of both sides of the foramen magnum. The medulla oblongata and cord and the inferior surface were pressed together as a result of herniation into the foramen magnum. The pressure was apparently greater on the left side than on the right.

Upon opening the paranasal sinuses the mucosa appears normal. There is a dehiscence, 4 mm. in diameter, apparently congenital, which connects the left lateral

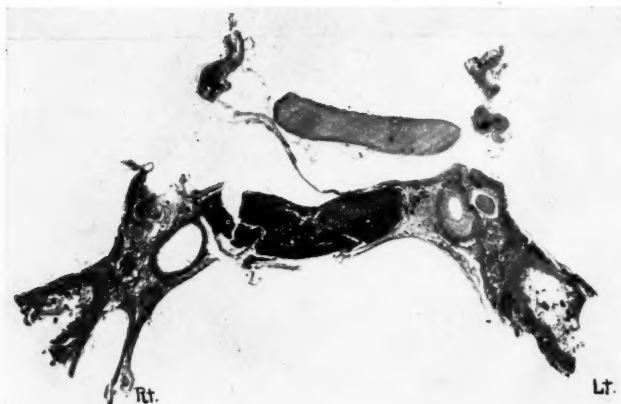


Fig. 7. Sections of left cavernous sinus, pituitary and right cavernous sinus, showing obliteration of all the venous channels around the nerves and left carotid artery, with an extensive obliterating endarteritis of the left carotid artery which was involved in the cavernous sinus inflammatory process. The contrast with the right carotid artery and sinus is marked.

sphenoid sinus with the sella turcica. On removing the right petrous bone about two ounces of pus escape from the abscess of the right jugular bulb and tissues of the pterygoid region. The right petrous pyramid appears cloudy. The left petrous bone appears normal.

The examination of the organs of the chest reveals an organized and granulating lower lobe of the right lung, with very little free pus. The upper lobe likewise shows some fibrosis and is partially aerated. There is a diffuse fibrous pleurisy present. The lower lobe of the left lung shows some induration with some congestion and edema, and the pleura is free.

The heart muscle and valves appear normal.

The liver shows some congestion and swelling.

The spleen is slightly enlarged and shows some fibrosis.

The kidneys show some engorgement, but the detailed outline is very distinct.

The remaining organs are not remarkable.

#### MICROSCOPIC.

Microscopic section of lung shows many of the alveoli filled with pus cells. There is edema and fibrinous exudate.

Other sections of the lung show congestion, some areas of hemorrhage. The bronchial tube contains some exudate. There is considerable aeration present in areas of the section. There is some fibrosis and inflammatory infiltration into certain areas.

Sections of the right lower lung show marked fibrosis, organizing inflammatory exudate, some areas of necrosis and hemorrhage and very little pus.

Microscopic sections of brain hernia show superficial necrosis and granulation. There are some areas of necrosis of the cortex.

Sections of the cerebral cortex show slight congestion and edema.

Sections of the medulla show edema.

Sections of liver show some central necrosis, large amount of fat, some perilobular fibrosis.

Sections of kidney show fibrotic glomeruli. Some show active inflammatory reaction. Many of the tubules show cellular degeneration.

Spleen shows engorgement and fibrosis, some thickening of the splenic cortex.

Heart shows some granular degeneration of the muscle fibers.

Microscopic sections of the left orbital tissue show the optic nerve with considerable inflammatory reaction and organized exudate in the sheath. The ophthalmic vein is completely thrombosed and organized, showing fibrous tissue and lymphocytes. Several of the smaller veins are likewise undergoing inflammation and organization. There is some inflammatory reaction in the muscles and around the nerves. There is some inflammatory reaction in the fatty tissues.

Microscopic section of the left cavernous sinus shows organization, fibrosis of the venous channels with considerable chronic inflammatory reaction around the third, fourth and sixth nerves and Gasserian ganglion. The internal carotid artery on this side shows a marked endarteritis with enormous thickening of the intima and almost complete obliteration of the lumen by fibrous and granulation tissue. The elastic lamina is broken and shows some calcium salts. The tissues, immediately around the pituitary, show some inflammatory reaction and granulation tissue, as does also the pituitary gland. The right carotid artery shows slight thickening of the intima. The venous channels are patent and there is slight increase in inflammatory reaction around the nerves.

Section of the left sphenoparietal vein shows some thrombosis.

Section of the superior longitudinal sinus, middle portion, shows some inflammatory reaction, some hemosiderin pigmentation. The posterior portion shows red thrombus with beginning organization.

Section of the right transverse sinus shows an organized thrombus with some inflammatory reaction, and numerous channels have been formed through the thrombus.

Right lateral sinus shows fibrous thrombus and numerous canalized blood vessels in the clot.

Sections of right and left inferior petrosal veins show fibrosis and thrombosis.

Section of connecting vein shows a septic thrombus with a small lumen containing pus. Immediately around the pus is a zone of granulation tissue in which there are canalized blood vessels, some newly formed venous channels and streptococci by special stain.

Section of anterior end of the straight sinus shows lumen filled with pus around which there is some granulation tissue and monocytic inflammatory reaction.

Section of the junction of straight and left transverse sinus shows a lumen filled with pus around which there is a considerable zone of granulations showing polyps and mononuclears, an increase in connective tissue and some newly formed blood channels.

Section of left lateral sinus near the bulb shows a lumen filled with pus surrounded by a rather thin zone of granulation tissue and a secondary zone of fibrous tissue. In the granulation tissue there are a large number of lymphocytes and many hydropic endothelial cells.

This case reveals an amazing amount of pathology in the venous channels of the brain without meningitis. Recovery was apparent when a lumbar puncture and a somewhat atypical structure of the foramen magnum produced a herniation and strangulation of the medulla oblongata, from which death rapidly followed.

The spread of infection from the middle ear through the bony structures of the petrous pyramid deserves a more thorough discussion than is possible in a brief presentation. These structures have recently received detailed investigation by Friesner, Eagleton, Kopetzky and Almour, and Ruskin. These authors have written clearly and convincingly upon petrositis and the literature upon the subject has been surveyed. Infections from the middle ear may spread to the petrous pyramid by way of perilabyrinthine or peritubular cells to the tip of the petrous bone or through the venous plexuses so abundant in this region. When the infection enters the petrous tip there may result an osteitis or an osteomyelitis with extensive accumulation of pus in the case of a pneumatized petrous bone. As a result of these pathologic processes, surgical and postmortem findings may reveal sequestration of bone, extradural abscesses on the upper surface of the petrosa, with perforation into the meninges. The pus may fill the entire tip of the petrous pyramid and involve the Gasserian ganglion, carotid canal and adjacent nerves. Inflammation in the adjacent structures to the petrous bone was recognized by Gradenigo in 1904, when he first described his characteristic syndrome. The infection may subside after a petrositis and the bone becomes filled with granulation or fibrous tissue and frequently newly formed bone results. In other instances, the pus may become absorbed or become inspissated and cheesy. Frequently a subdural abscess is formed in this region and it may rupture into the middle cranial fossa with the production of intracranial supuration. In other instances the infection spreads by means of the venous channels to the meninges. It is evident that cases of infection of the middle ear frequently involve the petrous pyramid by direct extension through the pneumatic cells, which are developed and intimately connected with the ear cleft and mastoid cells. A more careful observation at autopsy of our recent deaths from meningitis has convinced us that we have in the past overlooked the petrous bone as a focus of infection. This is easily understood, because a greatly diseased petrous bone may appear innocent upon gross inspection.

In the auditory apparatus there are natural avenues for the passage of infection to the intracranial structures. This refers to the

aqueduct of the cochlear with the perilymph fluid in the space between the bony and membranous labyrinth which offers a direct communication with the cerebrospinal fluid. The ductus endolymphaticus connecting the endolymphatic space of the labyrinth which terminates in the saccus between the layers of the dura where it covers the posterior wall of the petrous pyramid. When infection occurs in this duct it can easily extend directly through the deeper layers of the dura mater to the leptomeninges. This has undoubtedly been the pathway of infection in many cases of meningitis and brain abscess following suppurative disease of the auditory apparatus.

An extension of the perineural sheath into the labyrinth opens the way for the spread of infection from the inner ear to the posterior cranial fossa and is frequently the pathway of infection where meningitis complicates labyrinthitis. This sheath is likewise frequently involved, secondary to a leptomeningitis.

The pathways of infection of intranasal disease to the meninges are frequently easily demonstrated. This is not true, however, of infections in the auditory apparatus, which is understood because of the intimate association of the various complicated structures of the petrous portion of the temporal bone. For descriptive purposes, certain pathways have been discussed, but this does not imply that these pathways can always be demonstrated clinically, surgically or at autopsy. One receives an erroneous impression from the literature, which is inclined to describe the detached pathways that they can always be recognized clinically and pathologically, but it has been very difficult in many of our cases to indicate any one single avenue of infection to the intracranial complicating disease.

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THE PRESENT STATUS OF THE TREATMENT OF  
MENINGITIS.

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NEW YORK.

This part of the symposium is very hard to evaluate, as the results from the various types of treatment are very confusing and very unsatisfactory. Moreover, there is a very great diversity in our ideas as to what constitutes general meningitis and great confusion in the reports in the literature. Hence, at the outset, it is very important that we should all speak the same language when we discuss general meningitis, and the clarification of such a classification is the immediate important work to be done, as suggested by Dr. Tobey last year.

To digress a moment, the writer wishes to emphasize that his experience is the same as that of the previous speakers. He wishes to emphasize the points made by Dr. Williams and Dr. Neal as to the necessity for a thorough study of the bacteriology of meningitis. For over fifteen years we have been preaching that the so-called streptococcus mucosus capsulatus is, practically always, a pneumococcus. This is one of the points of confusion. Again, in reading the reports, we are bewildered by the results of the examination of the spinal fluid. Thus, one author reports a case of meningitis with a cloudy fluid but no organisms present. Again, we get reports that organisms were seen on direct smear but could not be cultivated. Again, that there were both Gram positive and Gram negative organisms in the same specimen. The reasons for such findings are well understood bacteriologically but are not usually appreciated clinically, hence the confusion.

A cloudy fluid, without organisms, does not necessarily mean generalized meningitis. The cloudiness may be due to an effusion of cells into the spinal fluid, the result of a tissue reaction due to organisms on the outside of the dura. There is an exact analogy in nature, well known to the ophthalmologist. In an ulcer of the cornea the

\*Presented as part of Symposium on Meningitis Secondary to Otitic or Sinus Infection before the annual meeting of the American Otological Society, Atlantic City, April 7, 1934.

anterior chamber of the eye is often full of pus (hypopyon). This hypopyon is usually sterile and the offending organism, the pneumococcus or streptococcus, is on the outer surface of the cornea; the tissue reaction is evidenced by the sterile pus (effusion of cells) in the anterior chamber. To the writer this explains the cloudy fluid without organisms.

The following points in the treatment are based upon an experience of twenty-seven years, in which we have seen 384 cases of general meningitis with sixteen recoveries. An analysis of these sixteen recoveries shows that two cases recovered without any treatment at all. In the others we used urotropin, sera and strained leucocytic extract or a combination of these. We could draw no conclusions. Dozens of the other patients received the same treatment and succumbed.

The methods of treatment may be classified as follows:

1. Surgical
2. Chemical
3. Serological
4. Physical
5. A combination of the above.

The surgical treatment may have two broad ends in view and may be subdivided as follows:

(1) Surgery to remove the original focus. (2) Surgical treatment of the meningitis. In our experience, the first is to be recommended, but the second is not. A fair analysis of the literature to date would lead one to the conclusion that all the surgical measures so far devised for the treatment of meningitis itself have been failures. An occasional patient gets well, but the vast majority do not.

Again, we cannot always remove the original focus and the observation narrows down this class. If there is a localized abscess or infection invading the meninges it is good surgery to remove it. However, some cases, especially the fulminating type, are due to direct invasion of the meninges, such as in injury to the dura, dislocation of the stapes, etc., and there is no time for a protective mechanism to act. Such cases follow a myringotomy or a direct invasion and are rapidly fatal. Such also occur in the removal of a polypus. Here the pathology is slightly different. In these cases the polypus has been walled off for years by a protecting wall of fibrous tissue, and if this is broken down we may get a rapid general meningitis. This has been observed in a considerable number of cases. The opposite is true in those cases that exhibit symptoms of menin-

gitis but only cloudy fluid. One such case, lately seen, had all the symptoms of a cerebellar abscess and meningitis—cloudy fluid but no organisms. An exploratory operation showed a sequestrum of the labyrinth. Removal of this sequestrum was followed by prompt recovery.

In the rapid forms there is no original focus that can be removed surgically. Again, we must remember that some of these cases are due to a sphenoid primarily, and others are due to an osteomyelitis of the base of the mastoid process and cannot be removed surgically in their entirety.

Looking upon repeated lumbar punctures as a surgical method we can say that some observers are impressed by this method; this is also true of drainage of the sylvian fissures and ventricles. These are all still subjudice.

There is rather a large class of chemical agents, such as the injection of urotropin, octogen, insulin, iodine, etc. All have been more or less discarded.

Among physical methods is the injection of the hypotonic salt solution intravenously with withdrawal of spinal fluid. Dr. Kubie will deal with this method.

The serological should be the ideal method of treatment, having in view the brilliant results obtained in the epidemic form of meningitis. Unfortunately, bacteriology has not advanced far enough in the work on pneumococcus or streptococcus to give us results. In the writer's opinion, the future method of treatment will lie in the serologic classification and treatment with surgical removal of the original focus if possible.

One thing can now be done by us all, and it will be a marked advance, and that is that we all agree upon the exact diagnosis of meningitis, and that the bacteriologic work is such that there is no doubt we are all talking about the same thing when we speak of general otitic meningitis.

57 WEST 57TH STREET.

LVI.

FORCED DRAINAGE FOR THE TREATMENT OF MENINGITIS SECONDARY TO EAR AND SINUS INFECTIONS.\*

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Two years ago the procedure of forced drainage was first described to this Society in a brief discussion of a paper by Dr. Philip Kerrison. Since that time a great deal of work has been done, and although we are still far from being in a position to indicate just how extensive or how limited is its therapeutic value, we welcome the opportunity to present our results as they stand before this same Society. In recent publications the theoretical background of the work has been brought together; and the methods of clinical procedure described. We will therefore review these two aspects of the method concisely.

Forced drainage, as a method of treating infections of the central nervous system, rests upon the basis of experimental work which was begun in 1925, at the point at which the classical work of Weed left off. Weed's work demonstrated that the volume of intracranial fluid, and hence the intensity of intracranial pressure, could be markedly altered by variations in the osmotic pressure of the blood. The experimental work upon which the method of forced drainage rests has added to these proved facts certain new data: namely, the varying origins of the cerebrospinal fluid which is newly formed when the osmotic pressure of the blood is lowered under varying intracranial pressures, the pathway along which that fluid will travel if given an opportunity to escape from the interstitial spaces of the brain, the possibilities of limiting the variations in intracranial pressure while altering the osmotic pressure of the blood, and the effect of these procedures upon normal animals and animals with meningitis. The problem may be briefly stated as follows:

1. Under normal circumstances (i. e., with an intracranial pressure that is higher than atmospheric pressure) practically all of the

\*Presented as part of Symposium on Meningitis Secondary to Otitic or Sinus Infections, before the annual meeting of the American Otological Society, Atlantic City, April 7, 1934.

cerebrospinal fluid is formed at the choroid plexus. On the basis of Starling's fundamental studies of the method of lymph formation, it is possible to prove that under conditions of normal or increased intracranial pressure all fluid which transudes through the arterial end of the capillaries throughout the central nervous system must be reabsorbed at the venous end of the capillaries, and therefore is not added to the main lake of cerebrospinal fluid which lies in the subarachnoid space.

2. When intracranial pressure is dropped to atmospheric pressure and kept there by continuous drainage, the cerebrospinal fluid is formed not only at the choroid plexus but by transudation through all of the small capillary vessels of the central nervous system. Under these conditions—that is, with reduced intracranial pressure, reabsorption at the venous end of the capillary system is reduced, and therefore the capillary transudate is not all reabsorbed but remains in the perivascular and perineuronal spaces and ultimately adds itself to the lake of the cerebrospinal fluid in the subarachnoid space.

3. Under these conditions, the fluid which is formed as a capillary transudate will move slowly along the perivascular spaces to emerge finally in the subarachnoid space.

4. The rate of formation of fluid as a capillary transudate can be remarkably accelerated by lowering the osmotic pressure of the blood, if this fluid is allowed to escape freely into the subarachnoid space by maintaining continuous drainage throughout the process.

5. If free escape is allowed for the newly formed cerebrospinal fluid, during the period in which the osmotic pressure of the blood is being lowered, there will be no material increase in intracranial pressure and no diffuse hydration of the substance of the central nervous system.

6. In the presence of inflammation of the central nervous system associated with cellular infiltration of the perivascular and perineuronal spaces, it has been possible to prove that this process of forced drainage actually washes much of the inflammation from the depths of the central nervous system to the subarachnoid space and out the path of drainage.

7. Both normal and sick animals have been subjected to this procedure repeatedly, and in no case have any injurious results followed. As clinical trials of the method are beginning to accumulate, it becomes evident that the method is equally safe for human beings.

8. There is presumptive indirect evidence, which has not yet been made direct and complete, that this procedure may increase the transfer of immune bodies from the blood to the cerebrospinal fluid.

9. It has been shown definitely that the best method of reducing the osmotic pressure of the blood is by carefully controlled intravenous injections of hypotonic saline solutions, which contain just enough salt to prevent hemolysis of the red blood cells. It has further been demonstrated that such injections, administered slowly, do not raise the venous pressure and do not increase the circulating blood volume, and therefore do not overload the circulation.

10. It is not too much to claim that this method of procedure is rational, safe and worth testing clinically in a very wide variety of infections of the central nervous system.

11. Forced spinal drainage differs from ordinary simple withdrawal of fluid from the subarachnoid space in the following fundamental respects: (a) The simple drainage allows the leptomeninges to collapse around the central axis of the nervous system and so to obstruct the natural pathways along which fluid moves, both in the interstitial spaces of the brain and in the subarachnoid space; (b) simple drainage merely removes the superficial lake of subarachnoid fluid and partially evacuates the ventricles, whereas forced spinal drainage makes it possible for the whole capillary bed of the central nervous system to participate in the processes of fluid formation, instead of leaving it confined to the specialized activity of the choroid plexus; (c) forced spinal drainage produces, therefore, a true perineuronal and perivascular drainage.

In the present state of our experience it is easier to speak of the contraindications than the indications for this procedure.

From the systemic point of view it is probably ill advised to pour hypotonic solutions into a patient who is suffering from an active pulmonary or upper respiratory tract infection. Although we have had no fatal accidents from this complication, we have on one or two occasions seen a patient made uncomfortable by an increasing outpouring of fluid from such inflamed areas. Similarly, it would be well to be cautious when dealing with a patient with any degree of cardiorenal deficiency. It is true that experiments would indicate that the method does not overload the circulation; but despite this fact, in dealing with human beings, caution is obviously desirable.

Despite the fact that our own records are deficient in this respect, it is of utmost importance to know before undertaking treatment whether or not there is an active blood stream infection. The old investigations of Weed and Wegefarth have recently been confirmed in the laboratory of Rich in the Department of Pathology at the Johns Hopkins Hospital, and would indicate that organisms can be carried over into the cerebrospinal fluid from the blood stream

by drainage of the fluid. On experimental grounds, therefore, this would seem to be an argument against using the method in the presence of a bacteremia. To what extent, however, this will limit its practical usefulness remains to be demonstrated. It is clear that repeated blood cultures should be made on all cases in whom this treatment is contemplated, before, during and after its use.

The outstanding contra-indication for the application of this procedure is the existence of any lesion into which fluid can pour and from which fluid cannot escape. Such a situation one encounters if there is an inflammatory obstruction blocking the aqueduct or the basal cisterns, or in brain abscesses, or possibly where one is dealing with an exudate which is too thick and gelatinous to make drainage possible. Proper laboratory studies and careful differential diagnoses must be made in order to rule out such conditions. One case is described below in which the symptoms of a cerebellar abscess were mistakenly interpreted as being the evidences of an oncoming meningitis. As a result, a sudden release of cerebrospinal fluid from the lumbar region and a very small intravenous injection (250 cc.) were enough to produce pressure conditions which caused respiratory failure and the death of the patient.

In the present state of our experience we can only say that it is our belief that, with the exception of the contra-indications already discussed, any infection of the central nervous system or its membranes can be attacked with this procedure. Furthermore, it is our experience that the earlier this is undertaken the better.

A special problem arises as to whether it is better first to clear out the foci of infection in the bone and subsequently treat the meningitis, or whether it may not be better to reverse the order of these procedures. Particularly, when dealing with a labyrinthitis, it may be well first to institute the drainage; then if the patient's meningeal condition begins to improve, to clear out the infected labyrinth while continuing the forced drainage both during and after the operation. These are points, however, which can be settled only by long experience and repeated trials.

Technical data have already been presented so fully in other places that only the essentials will be given here. The first purpose is to reduce intracranial pressure to atmospheric pressure as rapidly as possible without undue discomfort to the patient. With some patients it is possible to evacuate unhesitatingly all the fluids which will flow freely. With others, this withdrawal of the preformed lake of cerebrospinal fluid must be done very cautiously because of the production of headache and nausea. In comatose patients one can proceed



rapidly. With excited and delirious patients it is necessary to use sedatives. Sodium amytal intramuscularly, paraldehyde per rectum, and morphin and hyoscin have all proved effective.

When headache and nausea are too severe, it is necessary to begin the administration of hypotonic solutions almost at once. Otherwise one waits until the intracranial pressure has been thoroughly reduced before beginning the injection. Concentration of the injection solution varies from 0.45 per cent sodium chloride downward, depending upon the fragility of the red cells, as determined before treatment is begun. Fluid is administered in large volumes, varying from one to three liters in the course of from one to three hours.

The duration of drainage varies greatly. At times we have worked with short, repeated drainages of only one hour's duration; at other times continuing the drainage for several hours; and, on a few occasions, steadily for many days.

Throughout the treatment the patient reclines on his back on a specially constructed Bradford frame with a window opposite the lumbar region, and with his head several inches higher than his heels. It has not been possible always to use this method, and especially for shorter periods of drainage the ordinary posture is frequently employed. The frame, however, offers definite advantages and should be used whenever possible.

At times it is possible to increase the response to cerebrospinal fluid by decreasing the kidney output by intramuscular injections of pitressin or other pituitary extracts. In long continued treatments fluid is administered by every avenue that is available, but for short periods the intravenous method is the one of choice.

#### REPORT OF CASES.

We have treated too few patients to permit of any statistical analysis of our results. We are justified only in presenting our tentative impressions; and in order to present as fair a picture as possible we will describe first our failures.

CASE 1.—O. D., a large negro of 35 years, was seen at the Presbyterian Hospital on December 6, 1932. For a month the patient had suffered from mild headaches, earache, and tinnitus. Suddenly, the evening before his admission to the hospital, he became acutely ill, sank into a coma, and developed a temperature of 106°. The cerebrospinal fluid contained over 100,000 cells, with abundant pneumococci in the smears. His ear drums were bulging and were punctured, one yielding a rush of gas and the other bloody fluid.

Forced drainage was undertaken at once, and in two hours two liters of 0.45 per cent saline solution was injected intravenously, with a free drainage of over 200 cc. of cerebrospinal fluid. Two hours later the patient ceased breathing.



After his death the report on the blood culture showed an overwhelming blood-stream invasion with a pneumococcus, type 3, organism. At autopsy a fresh blood clot was found pressing on the cerebellum.

CASE 2.—A few months later an almost identical situation arose in a vigorous adult negro, in whom a rapidly progressive middle ear and mastoid infection gave rise to a severe meningeal inflammation. Again a type 3 pneumococcus was isolated, both from the cerebrospinal fluid and from the blood stream. Immediately after a radical mastoidectomy, the patient was subjected to forced drainage. The needle was retained for the larger part of two days, and during that time he was given repeated slow intravenous infusions of 0.45 per cent saline, with the drainage of several hundred cc. of cerebrospinal fluid.

For the first part of these two days of treatment the patient seemed to be improving. The intensity of the meningeal reaction lessened, and he became somewhat less comatose. The blood stream infection continued, however, unabated, and in the end the patient succumbed.

CASE 3.—Z. K., a 33 year old woman, who was admitted to the Holy Name Hospital in the care of Drs. Kennedy, Tennis, and later of Drs. Hare and Zabriskie. In this patient an otitis media was followed by a labyrinthitis and mastoiditis, which led to the performance of a mastoidectomy. During the course of these progressive stages of infection, a meningitis developed which later proved to be due to an untyped pneumococcus infection which was demonstrable both in smears and in cultures. One blood culture was taken and proved to be sterile.

Again the meningeal signs developed with great rapidity, with rapidly increasing coma and a temperature of  $105^{\circ}$ .

Treatment was begun within one day of the onset of the meningitis, and six very conservative drainages were given, each lasting only an hour, with the injection of from 250 to 1000 cc. of 0.5 per cent saline solution, and the recovery of between 55 and 110 cc. of cerebrospinal fluid.

Despite the conservatism of this treatment, there were marked changes in the cerebrospinal fluid findings. The cell count dropped from 4200 to 200 per cubic millimeter, and organisms could no longer be demonstrated in the smears. Nevertheless, despite the improvement in the fluid, her temperature remained elevated, the meningeal signs persisted, and the patient died six days after the operation, which was also six days after the first spinal drainage.

CASE 4.—To our list of fatalities we must add a brief reference to the one which we have already mentioned,—namely, the case in which a rightsided mastoiditis and mastoidectomy was followed by the development of a right cerebellar abscess. Here, unfortunately, the signs and symptoms simulated a meningitis so closely that the absence of any of the characteristic findings in the fluid was not sufficiently stressed. The patient was treated by forced drainage, and after the rapid sudden release of a large amount of cerebrospinal fluid, and the injection of only 200 cc. of 0.5 per cent saline, the patient died of respiratory failure. At autopsy the abscess was found to contain fluid which was under increased pressure. Apparently it had pressed downward to obstruct the aqueduct, and with the release of fluid from below, had caused a partial herniation through the foramen magnum, with the resulting interference with respiratory function.

This case needs to be mentioned only as a warning against the most serious danger which this procedure entails, namely that of pouring fluid into a lesion from which it cannot escape.

CASE 5.—A man in his middle 30's, was admitted to the Strong Memorial Hospital in Rochester, N. Y., with the symptoms and signs and the typical spinal fluid findings of a meningitis. It had originated in a middle ear infection, and the infection in the ear and in the cerebrospinal fluid proved to be that of a type 3 pneumococcus.

The patient was admitted to the hospital on the fourth or fifth day of the meningeal symptoms, and was subjected to a radical mastoid operation at once by Dr. Clyde Heatly. At that time the cell count was 2000 cells with a high fever, but a persistently negative blood culture.

The patient was placed upon a Bradford frame and subjected to forced drainage, with massive administrations of fluid, partly intravenously in the form of hypotonic saline, and partly with normal saline glucose solutions, and large volumes of tap water by mouth. The total volumes of fluid administered and of cerebrospinal fluid collected are not available. Gradually, however, the fluid became sterile, the cell count fell off rapidly, and at the end of the week the forced drainage was discontinued except for periodic tapping for a few days more. The clinical improvement from the institution of the drainage was rapid, and about three weeks from the onset of the illness the patient was allowed to return home.

A few weeks later, however, he developed a unilateral sixth nerve palsy, with some fever. He was readmitted to the hospital. He was operated upon again, and a small cholesteatoma, which had been overlooked at the initial operation, was removed from the wound. He was again subjected to forced drainage for a few days continuously, and again with rapid improvement. Upon returning home this time the patient remained well.

CASE 6.—J. V. D. was an 8 year old girl, who was admitted to the Manhattan Eye, Ear, Nose and Throat Hospital in the care of Drs. Cuning, Zabriskie and Hare. After three years of struggle with an ear infection, which had followed pneumonia, and which had necessitated repeated mastoid operations, the patient developed an intense meningeal reaction in the course of a week. Blood stream infection was never demonstrated, nor were organisms found in the cerebrospinal fluid; but the clinical course was typical for a severe, progressive meningitis, with high fever, stupor and symptoms of irritation. The fluid at first, although clear, coagulated rapidly; but with the rapid increase of meningeal symptoms it became turbid and contained 1600 cells, of which 60 per cent were polymorphonuclear.

The child was subjected to twenty forced drainages, which lasted from thirty to sixty minutes each. Fluid could not be introduced by vein, but was given by mouth and by nasal tubes, with the recovery of 30 to 60 cc. of cerebrospinal fluid each time.

During the course of the treatment the cell count dropped to 28, the protein became normal in amount, and there was a gradual disappearance of all meningeal signs and symptoms, except for a moderate degree of sensory and motor aphasia. With the sudden discharge of a large amount of purulent material from the wound, this healed over. The child has remained well for eight months since her discharge from the hospital.

Because of the incompleteness of our evidence as to the nature of the infection and the extent to which the meninges were directly involved in the infection, and because of the possibility that part of the picture might have been due to an epidural abscess, it must be admitted that this case is less impressive than the preceding one.

CASES 7 AND 8.—We have now two very characteristic cases to report: one is a child of 13 at the Manhattan Eye, Ear, Nose and Throat Hospital; the other is a child of 15 at St. Vincent's Hospital, Staten Island. In each case hemolytic streptococci have been recovered from the cerebrospinal fluid. In each case the meningitis followed shortly after a mastoiditis and mastoidectomy.

In one case, the infection in the ear had existed for almost a month, whereas in the other case it has been only of a few days' duration. In both, however, the meningitis developed rapidly in the course of a day, with characteristic signs and symptoms. The cerebrospinal fluid before treatment contained 4000 cells in the one, and 3900 cells in the other, with a marked predominance of polymorphonuclears and with positive smear and culture.

Forced drainages were instituted after one day of meningeal symptoms. One child received fourteen drainages in fourteen days, each lasting one hour; the other received five drainages. In the course of each drainage, between 1000 and 1500 cc. of 0.5 per cent saline was injected, and between 50 and 130 cc. of cerebrospinal fluid recovered.

In the one child the cell count dropped to 32, and in the other to 34; and in both the smears and cultures became negative. Furthermore, in both children the temperature dropped to normal, the symptoms and signs of meningitis disappeared, and an uncomplicated convalescence set in. Unfortunately in one child another physician made an ill-advised cisternal puncture, apparently producing an injury to the medulla, with bleeding and a prompt rise in temperature. This child suffered a set-back, from which it is now recovering.

CASES 9 AND 10.—Finally we have to report two cases in which an intense meningeal reaction, without complete evidence of infection, appeared suddenly in patients who had suffered for years from chronic ear or sinus infection. In each case the intoxication was profound and alarming, and the response to treatment complicated by the uncertainty as to the exact nature and extent of the bony involvement.

CASE 9.—H. E. M., age 22, a patient of Drs. Page, Hare and Zabriskie, in the Manhattan Eye, Ear and Throat Hospital. After twelve years of chronic middle ear infection, the patient developed an acute exacerbation with a rapid outcropping of meningeal signs and symptoms, and then a mastoiditis and labyrinthitis. Superimposed upon the symptoms of the mastoiditis and labyrinthitis were evidences of increasing intracranial pressure, and shifting signs and symptoms of various focal injuries to the brain as well. The cerebrospinal fluid at this stage was under high pressure, with 770 cells of which 60 per cent were polymorphonuclears. A mastoidectomy and labyrinthectomy were performed, and resulted in a temporary improvement and a temporary reduction in the number of cells in the cerebrospinal fluid to 275. Organisms were never found, either in the blood stream or in the cerebrospinal fluid.

Four days after the onset of the meningeal symptoms, forced drainages were undertaken. Five treatments were given, of one hour's duration each, with the administration of 1000 cc. of 0.5 per cent saline, and the recovery of between 80 to 100 cc. of cerebrospinal fluid.

During the very first drainage the cell count dropped from 950 to 360; but during subsequent drainages the cell count rose as high as 1800, and then finally fell until after the last treatment it had dropped to 100, with a diminution in the excess of globulin and protein.

The clinical course of the patient was somewhat stormy. There were further transient local weaknesses and fluctuations in his consciousness. Gradually, however, after the termination of the series of drainages, the temperature dropped, almost all local signs disappeared, and he was conscious and oriented. This improvement continued steadily, and now eight months after discharge from the hospital he remains well.

CASE 10.—The last patient, 52 years of age, was also at the Manhattan Eye, Ear and Throat Hospital, in the care of Drs. Hare and Zabriskie. In this patient the meningeal reaction followed several years of a right frontal sinusitis, with gradual erosion of the posterior sinus wall. The meningeal symptoms developed rapidly in the course of two days, with high fever, incontinence and a rigid neck. Organisms were never recovered either from the blood stream or from the cerebrospinal fluid. The fluid, however, before treatment, contained 2500 cells, of which 95 per cent were polymorphonuclear, and had a slightly yellowish tinge.

Six drainages were given of one hour each in six days, with the injection of between 1000 and 1500 cc. of salt solution, and the recovery of between 50 and 100 cc. of cerebrospinal fluid. During the course of these treatments the cell count dropped to 275, the fluid becoming clearer but remaining yellow tinged. The improvement during the treatment was not steady. There was an improvement for a few days, followed by a relapse into coma, with a high temperature and a suspicion of pneumonia. As a result, treatment was stopped, but without further intervention improvement again set in. After a short interval the toxemia overwhelmed the patient, who began to have periods of cyanosis and apparent heart failure, in one of which she died.

#### COMMENT.

From these ten cases no conclusions may be drawn as to the influence which the introduction of this treatment might have upon the mortality rate in this type of infection. Individual cases have shown remarkable apparent responses to the procedure. Such episodes, however, are notoriously deceptive and can be trusted only when backed by extensive experience and careful statistical comparison with the results of other methods of treatment. Obviously the results will vary with the virulence and quantity of the infection, with the extent of the bony involvement, with the nature of the organisms, with the presence or absence of blood stream infection, with the general intoxication of the patient, and the adequacy of the response of all of the organ systems. The specific treatment of the meningitis alone is only one part of the struggle, and no form of treatment which is directed towards this alone can possibly save all patients. We are prepared to claim only that our experiences thus far justify us in pushing further along these same lines.

It is to be hoped that with increasing utilization of the method our form of attack will become more consistent. Up to the present we have varied from short periods of forced drainage, frequently repeated, to long periods stretching over several days. Unfortunately

all approaches to the central nervous system present special technical problems, and we cannot say that all of these have as yet been overcome. Nor have we as yet solved the fundamental chemical problem as to the optimal method of reducing the osmotic pressure of the blood and thus stimulating, maximally, the new formation of perivascular transudate. These are aspects of the problem which will have to be presented at a later time.

34 EAST 75TH STREET.

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LVII.

PATHOLOGY AND ROUTES OF INFECTION IN LABY-  
RINTHITIS SECONDARY TO MIDDLE EAR OTITIS.\*

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The material presented in this paper is compiled from two fields of study. The microscopic observations were made by one of us, D. Wolff. The clinical observations were made by the other, L. W. Dean.

The microscopic investigations are based upon forty-six cases of otitis media and an examination of serial sections from sixty-eight ears. Both ears in each of the forty-six cases could not be examined. Bilateral observations were made in twenty-one cases. In the bilateral group studied evidence of otitis existed in each ear, although frequently one side was more fulminating than the other. Suppurative labyrinthitis was observed in fourteen ears (eleven cases).

The sections show that infection may pass from the middle ear to the labyrinth by direct extension and by indirect extension. The routes of direct extension are:

1. Fistula through the round window.
2. Fistula through the oval window.
3. Fistula through the bony capsule as a result of
  - (a) Necrosis of bone or
  - (b) Mechanical trauma.

Indirect extension is by the blood vascular route and lymph vascular route.

It is, of course, quite impossible to classify definitely the processes of nature. We must, therefore, recognize that no one route excludes another and the infection may be a combination of the direct and indirect extension.

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\*From the Oscar Johnson Institute, Washington University Medical School, St. Louis, Mo. This work was made possible by the Ball Research Fund.

Presented as part of a Symposium on Labyrinthitis Secondary to Middle Ear and Mastoid Infections before the annual meeting of the American Otological Society, Atlantic City, April, 1934.

## DIRECT EXTENSION.

*Fistula Through the Round Window.*—The frequency with which the round window may act as a route of extension from the middle ear to the labyrinth is dependent upon certain anatomic features. First among these may be considered the location of the window; second, the structure of the membrane covering it.

**Location:** The round window is deeply placed in a niche, the fossula fenestræ cochleæ, within the overhanging rim of the promontory. The dimensions of the niche are approximately 1 mm. in depth by 2 mm. in diameter. This niche is even more completely hemmed in by the presence of the subiculum, a curved bony bar or ridge which supports the promontory and separates the niche from the sinus tympani. Once infection gets started in the middle ear a mass of pus collecting in the niche drains from it with difficulty.

The close proximity of the sinus tympani, another deep pocket in which pus is apt to become collected, is an important anatomic point to be considered in the pathology of the round window. The sinus tympani lies above and posterior to the niche of the round window, in fact, in just the position to pour its suppurative contents into the niche of the round window if the patient is in the upright position, while the niche of the round window could conceivably pour its contents into the sinus tympani if the patient is reclining.

In infants there is, in addition to the sinus tympani, another pus-holding depression. This is the fossula of Hyrtl or the petrosal fossula. It is a somewhat funnel-shaped fossula and runs parallel to the aqueductus cochleæ. In the very young infant its developmental stage is sometimes of great significance when considering pathologic routes to the dura. For the present this point does not concern us. But the proximity of the pocket to the niche of the round window is significant in studying suppuration of the region.

The niche of the round window and the sinus tympani are both sites where remnants of mesenchyme are slow to be resorbed in infant ears. Tags of mesenchyme may hang suspended on the membrana tympani secundaria (Fig. 5) or partially fill one angle of the niche, thus interfering with easy drainage. These tags may themselves become infected. As previously reported by Wolff, mesenchymal remnants may persist until the infant is 13 months of age.

**Structure of membrana tympani secundaria:** This organ is not a simple round membrane stretched evenly across the aperture of the



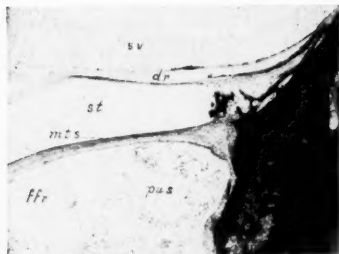


Fig. 1.

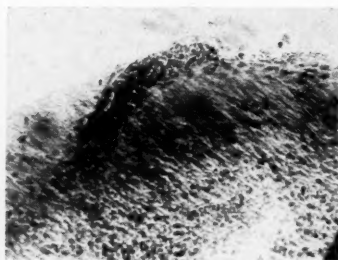


Fig. 3.



Fig. 2.

Fig. 1 (1). Very early infiltration of the round window membrane. Mucosal cells toward the cut end of the membrane have been entirely sloughed off.

Fig. 2 (4078). The arrow lies in the niche of the round window which is filled with pus. It points to the membrane which shows dissolution and pus is entering scala tympani of the cochlea. Niche of oval window is also filled with pus—5 months.

Fig. 3 (4334). A blood vessel crossing the fibrous layer of the round window membrane. Age, 19 days.



fenestra rotunda. On the contrary, it is drawn into the labyrinth far enough to be attached to the basal portion of the bony spiral lamina of the cochlea. This causes the membrane to present a concave surface to the middle ear and to pass through three planes in space. The pulling in of the membrane deepens the niche of the round window, increasing its efficiency as a pus-pocket. The membrane is sufficiently elastic so that if pressure is increased within the labyrinth it may bulge out into the middle ear.

As measured in the sections, the membrane averages approximately .065 mm. in thickness. Like the drum membrane, it is composed of three layers. These are a mucosal layer, a fibrous layer and a labyrinthine lining. (Fig. 1.) The mucosal is a continuation of the mucosa of the middle ear. On the membrane, however, the cells become flattened to low cuboidal, almost squamous type. Minute capillaries occur in the relatively thin submucosa of these cells. In five instances in the present study these vessels have been traced across the fibrous layer, showing that vascular channels do pass from the middle ear over to the labyrinthine side. (Fig. 3.) These observations were made on only every tenth section. If every section were read the vesels would probably be found constantly.

The fibrous layer of the membrane is formed from the periosteum of the bone to which the membrane is attached. It is much more impervious to infection than the mucosal layer, as noted below. The labyrinthine layer is difficult to demonstrate and is scarcely more than the inner layer of the fibrous, since it too is formed from the periosteum (Ruttin) of the labyrinth, or endosteum, as we shall term it. Because of the one free surface, the nuclei of this layer are more rounded than those of the fibrous layer. The three layers are fairly well shown in figure 1.

The frequency of a pathologic condition in the region of the round window is shown by the fact that of the sixty-eight ears examined sixty-four showed pus in the niche of the round window. Infiltration of the mucosal layer of the membrane was observed in forty-three ears. Infiltration of the fibrous layer occurred in twenty-five ears. Simple edema of the fibrous layer was observed in six additional ears. Complete breaking down of the round window was observed in only four ears, each in a different case. In the twenty-one bilateral cases observed both niches of the round windows contained pus in all except two cases. One of these patients had a very slight otitis and pus did not occur in either niche. In the other case pus was found in the left side only.

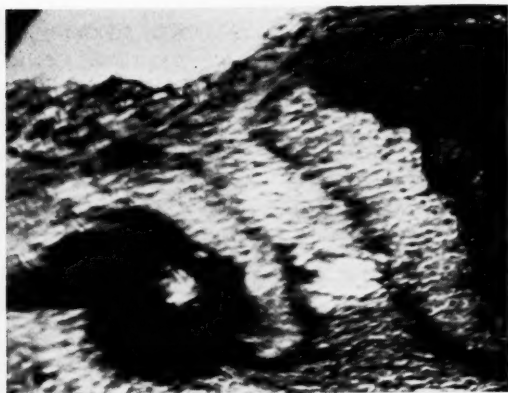


Fig. 4.



Fig. 5.

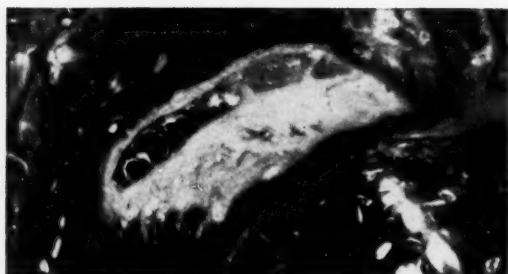


Fig. 6.

Fig. 4 (4078). Edema of the fibers of the annular ligament. Age, 5 months.

Fig. 5 (4297). A mesenchymal tag suspended on the round window membrane. Such tags may block drainage or become infected themselves. Age, 3½ months.

Fig. 6 (5115). Healing in the perilymphatic space of a canal leaving a supuration in the endolymphatic canal. Age, 5 years.

Early infiltration of the membrana tympani secundaria shows figure 1 with scattered pus cells in the niche. Infiltration of the mucosal layer is present along the region of the origin of the membrane. Toward the middle of the membrane the mucosal cells have been entirely sloughed off, indicating the advisability of getting good drainage as early as can be done with safety in acute middle ear otitis. The pathologic changes in the round window are best prevented by thorough middle ear drainage. In figure 2 the round window is undergoing complete dissolution. The stapediovestibular joint is also involved, illustrating the point that the two routes are not mutually exclusive. Between these stages is a phase not illustrated. We have stated that simple edema of the fibrous layer was observed in six cases. This is evidenced by clear spaces, either elongated or rounded, in the midst of the fibers. Such spaces cannot be considered artefacts if they occur only in this membrane and not elsewhere in the tissue. The edema is probably induced by toxins given off by the bacteria present. If it exists long enough it softens the durable fibrous elements of the middle layer, breaking down the natural resistance of this layer and permitting infiltration of the pus cells from the submucosa. The sections show, however, that the fibrous layer is much more resistant to infiltration than is the mucosal. The statistics here presented show that for this group it is more than twice as resistant.

Comparison of human with certain animal material (white rat and rabbit) leads us to believe that the resistance of the fibrous layer of the human ear is relatively much greater than in the animals cited. If this be true, statistics regarding the frequency of round window fistulæ in induced middle ear disease and labyrinthitis in experimental animals of these species give little clue for conditions in human ears.

*Fistula-Oval Window.*—Anatomically the niche of the oval window, fossula fenestræ vestibuli, is in a more exposed position than that of the round window. Paracentesis therefore drains it more readily. This probably explains why pus was found less frequently in this fossula than that of the round window. The stapes articulates within the oval window, being held in place by the fibrous annular ligament, which may really be said to be a double layer of fibers forming a typical joint-capsule. No membrane exists on the inner side of the stapes other than the endosteum covering the rest of the wall of the labyrinth. When the fibers of the annular ligament are torn or broken down by disease the labyrinth is immediately exposed to the middle ear at that point. As in the case of the round window, pathologic changes of the round ligament are best prevented by thorough middle ear drainage.



Fig. 7.

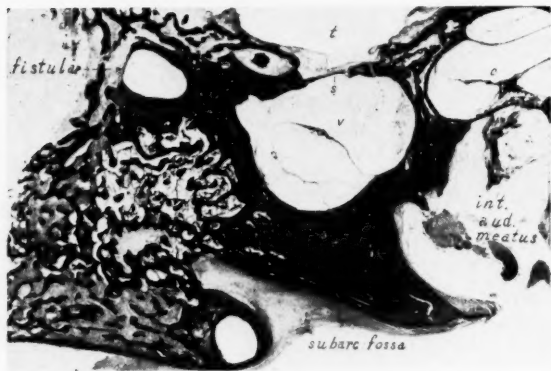


Fig. 8.

Fig. 7 (4336). Fistula through oval window. Footplate of stapes is outlined in white. The arrow indicates pathologic tissue which has dislodged the anterior end of the stapes. Age, 12 years.

Fig. 8 (3865). Fistula of horizontal canal in congenital syphilis. Note diseased condition of perilyabyrinthine marrow. Age, 4 months.

In the study of the oval window it was found necessary to discard eight cases because of trauma at autopsy, thus making sixty the number observed. Pus was observed in the niche of the oval window in fifty-one ears. Obvious fistulae through the stapediovestibular joint capsule were present in four ears. This makes the percentage of fistulae by this route greater than by the round window in the cases here considered. In practice fistulae of the oval window are found at operation more often than fistulae of the round window. Simple edema of the joint ligament was observed in three ears. Figure 7 shows a typical fistula of the stapes where the inflammatory tissue

has broken down the joint-capsule and dislodged the stapes. The place of the capsule is being filled with scar tissue. Edema, as in the round window, is manifested by empty spaces appearing in the fibrous tissue. Such a condition in combination with infiltration of the joint is seen in figure 4.

*Fistula, Bony Capsule, Necrosis.*—Fistulae of the bony capsule as a result of necrosis occur most frequently in the horizontal canal and in the promontory. Figure 8 shows a fistula of the horizontal canal in a case of congenital syphilis. This occurred in a 4 months' white child who came into the hospital with a history of fever, vomiting and occasional convulsions. The sections show that the bony wall has been eroded but the canal has not yet been invaded. Numerous osteoclasts occur along the border of the lesion. Diseased bone marrow is present. This case illustrates the fact that we can have symptoms of labyrinthitis with fistula in which no surgical procedure is indicated. Any surgical procedure for this middle ear otitis with labyrinthitis, performed before the specific process has been controlled so far as possible, would only result in the breaking down of nature's barriers and in rapid extension of the osteomyelitis. The treatment in this instance is systemic.

If such a fistula appeared in a simple chronic suppurative otitis, uncomplicated by syphilis or tuberculosis, with or without cholesteatoma, it would not in itself be an indication for operation on the labyrinth. The suppurative process may be walled off and a satisfactory result secured through mastoid operation.

Chronic necrosing osteitis and cholesteatoma invade the capsule very slowly. Figure 15, No. 3956, taken from a case previously reported in the literature (Birsner-Wolff), shows how completely walled off the cholesteatoma is. This case had a running ear for eight and one-half years. The exact age of the cholesteatoma we do not know, but it never did invade the labyrinth. The damage was done by its backing the pus into the antrum and middle ear, thus preventing drainage upon paracentesis. Cholesteatoma and chronic necrosing osteitis invade the labyrinth capsule very slowly. Nature gives opportunity for walling off the infection when it attacks the labyrinth. Even if the labyrinth is completely destroyed, in these chronic processes the destructoin is so slow that the walling-off process advances before the infection so that meningitis is not often the result.

Acute diffuse labyrinthitis is the result of an acute otitis media or of an acute exacerbation of a chronic otitis media.

Any infant having an infected ear and vomiting should be a suspected case of labyrinthitis. Infants cannot complain of vertigo

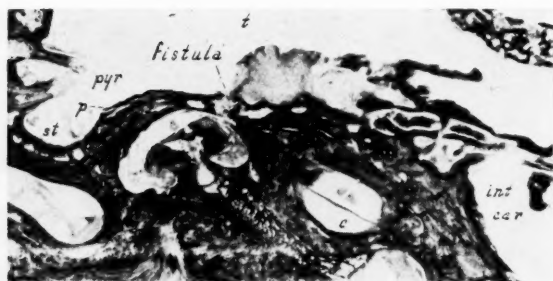


Fig. 9.



Fig. 10.

Fig. 9 (22). Fistula through bony capsule into promontory. This level is above the niche of round window, but sinus tympani and the pyramid of the stapedius muscle are clearly seen. Age, 5 years.

Fig. 10 (22). Fistula through bony capsule into horizontal canal. Note new-formed blood vessels in ampulla. Age, 5 years.

or deafness. Nystagmus in infants with labyrinthitis is only rarely observed. It would not be present before ocular fixation has developed. The caloric test gives valuable information. In using water at 68 degrees, if the labyrinth is dead, reactions indicating this condition will be present. Water at 68 degrees used in the ear of an infant with normal labyrinths causes nystagmus if fixation has developed, and a pulling of the eyes toward the ear tested if fixation is not present. Usually in infants under the age of 7 months with a normal labyrinth the caloric test, using water at 68 degrees, causes the eyes to deviate toward the tested side. Rarely at the age of 4 months, instead of the drawing of the eyes, a labyrinthine nystagmus directed to the opposite side appears. When this does occur the explanation of it is that ocular fixation has developed early. Oculists do not agree as to the age at which fixation is developed.

Fistula of the promontory through necrotic bone is illustrated in figure 9, No. 22. At radical mastoid operation this promontory

was found necrotic but no deeper entry was made except for "light curetting." The case history is given below.

*Fistula, Bony Capsule, Mechanical Trauma.*—A fistula produced by mechanical trauma in operative procedure in an attempt to prevent meningitis arising from a known labyrinthitis is seen in figure 10, specimen 22. This is a 5-year-old child with a case of neglected otitis media. A left mastoidectomy was done and a week later a right mastoidectomy. Eight days later it was found necessary to do a left radical mastoid operation, whereupon partially necrotic bone was removed from the horizontal semicircular canal. The region of the promontory was found necrotic but no deeper entry was made except for light curetting. One month later the patient died. Autopsy showed left temporal lobe abscess and left cerebellar abscess with basilar meningitis. Figure 10 shows the newly formed blood vessels in the ampulla of the horizontal canal in the attempt to heal. Just below the ampulla the marked necrosis of the capsule is evident.

Figure 6 shows Nature's method of healing in another labyrinthine operation in a post-scarlet case (No. 5115), to be reported in the literature (Cone-Wolff). Here we see scar tissue completely filling in the perilymphatic space, leaving a suppurative condition in the endolymphatic channel.

From the clinical point of view, labyrinthotomy of one type or another is sufficient but good drainage must be established. With good drainage the labyrinth will be obliterated. Labyrinthectomy should never be done unless there is evidence of meningeal involvement. The objections to labyrinthotomy, as shown in these specimens, is the fact that in the production of fibrous tissue and new bone, a pocket of pus may be walled off and remain a menace to the individual. An apparently healed suppurative labyrinthitis may produce meningitis because of such a residual pocket.

In labyrinthotomy one confines his surgical work to the semicircular canals, removing the bone between the oval and round window, and taking away just the shell of the promontory, being careful not to disturb the organized protective tissue and being particularly careful not to traumatize the modiolus.

There is little danger of a walled off circumscribed labyrinthitis causing a meningitis. If circumscribed labyrinthitis progresses and we have a chronic diffuse labyrinthitis develop the clinical problem becomes immediately a more serious one. A fistula discharging pus



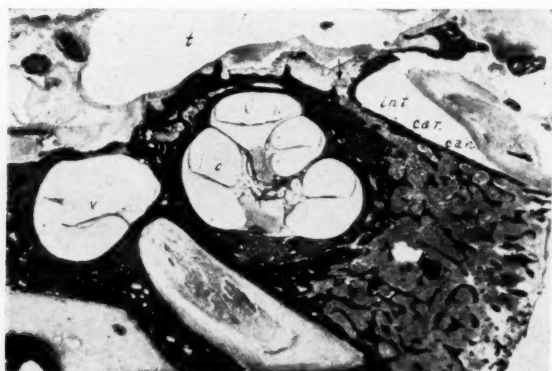


Fig. 11.

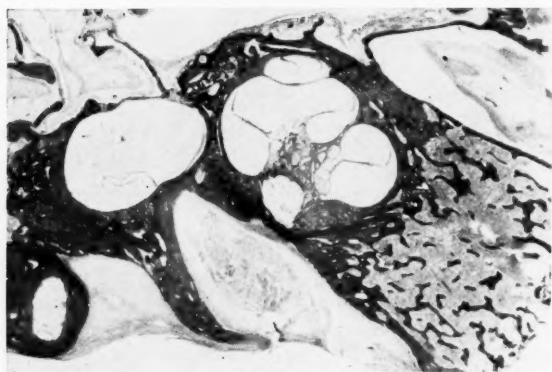


Fig. 12.

Fig. 11 (4674, Sec. 284). The pattern of marrow spaces in the petrous apex adjoining the cochlea. The arrow indicates direct communication with the submucosa of the middle ear. Age,  $3\frac{1}{2}$  months.

Fig. 12 (4674, Sec. 250). The pattern at a higher level. Note the direct communication with the Haversian systems surrounding the cochlea.

with a dead labyrinth suggests chronic diffuse suppurative labyrinthitis. A fistula discharging pus with labyrinthine function present, whether it is in the promontory or horizontal canal, suggests circumscribed labyrinthitis.

The effort on the part of Nature to limit the infection must be respected. The slightest trauma of the labyrinthine capsule or even a jar is certain to break down these beginning delicate barriers. The use of a gouge in a mastoid operation would have a deleterious effect. From the pathologic standpoint it is certain that operations should



not be done on the labyrinth in acute diffuse suppurative labyrinthitis unless there is evidence of a beginning meningitis. In the earliest stages it is better to postpone, if possible, the mastoid operation and when you do operate, avoid any instrument that may cause a jar. If and when the labyrinthitis becomes chronic a labyrinthotomy can be done in safety.

Another reason for not operating for a suspected case of diffuse suppurative labyrinthitis is that the lesion may be a diffuse serous condition and not a suppurative process. Diffuse serous labyrinthitis may involve all the structures of the labyrinth with complete deafness and total loss of labyrinthine function. The difference between the diffuse suppurative and diffuse serous labyrinthitis is that in the serous labyrinthitis there will be some return of the function. It is axiomatic that because of the early destruction of the soft tissues in diffuse suppurative labyrinthitis there can be no residual labyrinthine function. If there is a remnant of function, cochlear or vestibular, it is not a diffuse suppurative labyrinthitis. It is evident that when there is a remnant of hearing or labyrinthine response to stimulation the lesion is a circumscribed labyrinthitis or a diffuse serous labyrinthitis, and an operation on the labyrinth is not indicated.

In this study photographs have not been included which depict serous labyrinthitis, although we recognize and classify such conditions in the laboratory. In our records we have described the condition in 3956 as serous labyrinthitis. The difficulty of distinguishing between postmortem and agonal alterations, which might produce the microscopic picture of a serous labyrinthitis, are admitted. We frequently see under the microscope a pink-staining, clear serous fluid within the labyrinth. We also see marked edema of the perilymphatic tissue such as that illustrated in figure 14, where this does not seem to be solely due to artefact of technic. Bowing or sagging of Reissner's membrane, also a distorted and excessively liquefied state of tectorium, are points to be considered. When marked edema of the round window membrane or round ligament exists it is conceivable that a concomitant chemical alteration of the labyrinthine fluids may occur, thus producing a serous labyrinthitis.

#### INDIRECT EXTENSION.

*Blood, Vascular Route.*—In studying the microscopic anatomy of the temporal bone many possibilities present themselves for routes of extension from the middle ear by way of the blood vascular route. The presence of capillaries crossing the fibrous layer of the round window membrane has already been cited in this paper. (Fig. 3.)

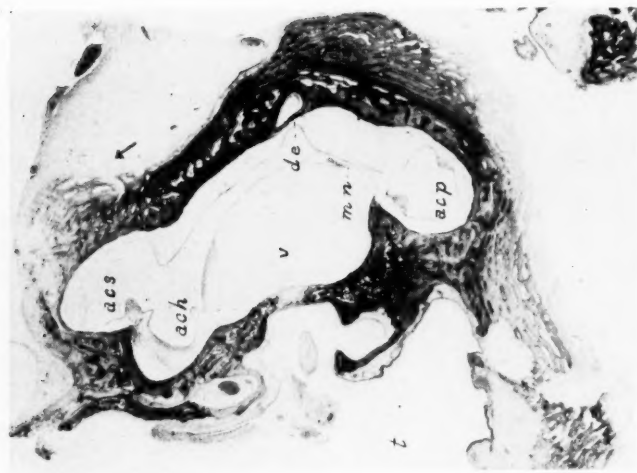


Fig. 14 (4334). Note the pattern of marrow spaces and Haversian systems surrounding the vestibular labyrinth. The arrow indicates a communicating canal entering the dura. Perilymphatic tissue is abnormally distended. Age, 19 days.

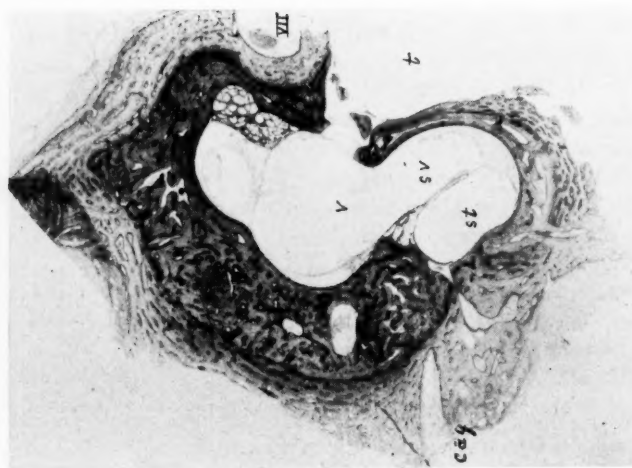


Fig. 13 (1). The vein accompanying the cochlear aqueduct does not follow the aqueduct but leads off in a separate canal which ends in a marrow space. Infant.



Fig. 15 (3956). Cholesteatoma in the middle ear. The arrow indicates perforation through which stratified squamous epithelium entered tympanum. The cholesteatoma is well walled off by the epithelium. Niche of oval window is filled with scar tissue. Age, 10½ years.

The extensive bed of marrow and Haversian systems surrounding the otic capsule, particularly of the infant, must be fully appreciated. This condition is well illustrated in the 3½-months infant, figures 11 and 12. In figure 11 the marrow spaces are in direct communication, as indicated by the arrow, with the middle ear offering a route of direct extension for developing osteomyelitis. In figure 12 we see how the marrow spaces narrow down to the Haversian systems which intimately surround the cochlea. Some of these Haversian canals have been traced to the endosteum of the labyrinth. Figure 13 illustrates a canal leading from a perilabyrinthine marrow space to the channel of the blood vessel accompanying the cochlear aqueduct. Blood vessels have frequently been observed passing from the mucosa of the middle ear into the Haversian systems of the bony capsule.

In figure 14, No. 4334, the arrow indicates a point where the lymph spaces of the dura and the marrow spaces of the labyrinthine capsule communicate. Similar areas can be picked out in figure No. 1. The marrow in these specimens is essentially normal, but note the condition of the marrow in figure 8, congenital syphilis.

The intimate relationship between the marrow spaces in the petrosa and the endosteum of the labyrinth demonstrates the danger of neglecting infection in these structures. The best way to pre-

vent suppurative labyrinthitis is to operate and drain at a proper time neighboring bone areas when they are infected.

The best treatment of labyrinthitis is its prevention. This can only be accomplished by adequate drainage, at the proper time, of an infected middle ear and infected marrow spaces adjacent to the endosteum of the labyrinth. (Figs. 11 and 12.) Unfortunately, if done at the wrong time, such a procedure will have a tendency to produce rather than to prevent labyrinthitis. For instance, if we have an early progressive, virulent middle ear otitis with labyrinthitis and meningitis, operation will only do harm rather than good because the body is not capable of limiting this process, and traumatism only hastens the spread of infection. In such a case one must depend on other methods for handling the meningitis rather than operation on the mastoid. In the same way operation for middle ear infection in the early stages hastens the production of labyrinthitis. As soon as the clinical picture indicates that Nature has built a barrier to the infection, then drainage of the infected area without injury to the limiting process is the best prevention of labyrinthitis.

It is equally evident that surgical neglect of chronic necrosing mastoiditis or mastoiditis with cholesteatoma is liable to cause labyrinthitis. Such processes are well walled off but are slowly progressing. (Fig. 15, No. 3956.) They can be operated on with safety.

It requires the very best clinical judgment to determine just when an acute otitis has reached the stage which justifies the surgical attack on the middle ear otitis and osteitis for the purpose of preventing labyrinthitis.

In considering the clinical significance of these labyrinthine lesions we must remember that in middle ear otitis, particularly in the so-called acute serous catarrh of the middle ear, we can have evidences of labyrinthine involvement without actual invasion of the labyrinth. That is, in acute serous catarrh of the middle ear we can have decreased hearing, nystagmus, subjective vertigo, which is so promptly removed by the drainage of the tympanic cavity, either by opening the eustachian tube or by myringotomy, that it precludes the diagnosis of either infection or serous labyrinthitis.

KEY TO ABBREVIATIONS.

<i>acl</i>	ampulla canalis lateralis (horizontal semicircular canal)
<i>acp</i>	ampulla canalis posterior
<i>acs</i>	ampulla canalis superior
<i>c</i>	cochlea
<i>cc</i>	crus commune
<i>caq</i>	cochlear aqueduct
<i>car</i>	carotid
<i>cp</i>	canalis posterior
<i>de</i>	ductus endolymphaticus
<i>dr</i>	ductus reuniens
<i>et</i>	eustachian tube
<i>ffr</i>	fossula fenestrae rotundae (niche of round window)
<i>m</i>	malleus
<i>me</i>	mesenchyme
<i>mn</i>	macula neglecta
<i>mt</i>	membrana tympani
<i>mts</i>	membrana tympani secundaria (round window membrane)
<i>p</i>	promontory
<i>pyr</i>	pyramid
<i>s</i>	stapes
<i>st</i>	scala tympani
<i>su</i>	subiculum
<i>sv</i>	scala vestibuli
<i>t</i>	tympanum
<i>v</i>	vestibule
<i>VII</i>	Facial Nerve

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## LVIII.

### DIFFERENTIAL DIAGNOSIS IN LABYRINTHITIS SECONDARY TO OTITIC INFECTIONS.\*

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#### INTRODUCTION.

It has been customary to classify labyrinthitis as circumscribed, fibrinous, serous and purulent. Only the last named type carries with it a threat to life. From an etiologic standpoint, there are types of labyrinthine invasion which are concomitant to mumps, scarlet fever and other toxemias which are not germane to this discussion. Those labyrinthine lesions which occur as a reaction to tympanomastoid infections only are the topic of this symposium. Among these the question heretofore has concerned itself primarily with assembling clinical and other diagnostic data to establish

(1) a labyrinthine lesion

(2) to differentiate it from some other intracranial lesion whose symptoms and laboratory findings simulate the picture a labyrinthitis usually presents, and

(3) having determined that the lesion was located in the labyrinth to effect a differentiation between the serous and the diffuse purulent types.

In the recent advances made by otology there has developed clinically a better comprehension of all the lesions which are located in the petrosal pyramid of the temporal bone. Labyrinthine lesions are only one set of these. It is now necessary to deal with lesions located on the outside of the labyrinthine capsule, lesions lodged in the osseous structure surrounding the labyrinth—that is, the perilabyrinthine regions. Perilabyrinthitis presents definite clinical and objective symptoms, which have connotations not comprehended heretofore. They very definitely constitute a threat to life, in addition to the dangers inherent to a purulent diffuse labyrinthitis. In dealing with

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\*Presented as part of a Symposium on Labyrinthitis Secondary to Middle Ear and Mastoid Infections before the annual meeting of the American Otological Society, Atlantic City, April, 1934.

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questions of differential diagnosis it is necessary to distinguish between the following lesions:

- (a) perilabyrinthitis
- (b) serofibrinous labyrinthitis
- (c) hematogenous labyrinthitis
- (d) labyrinthine fistula
- (e) purulent labyrinthitis
- (f) cerebellar abscess
- (g) localized meningitis.

Differential diagnosis of labyrinthitis, secondary to otitic suppurations, to be of more than academic interest must seek to establish a guide for the type of therapy needed to cure the lesion, and in what is here presented, an effort is made to keep that objective constantly in view.

#### DIAGNOSTIC DATA.

Upon pathologic examination of cases of acute and chronic labyrinthitis which come to section, various subdivisions of the lesion may be established. Among such divisions are serofibrinous labyrinthitis with, and also without endolymphatic hydrops; circumscribed serous and circumscribed purulent labyrinthitis; and hydrops labyrinthi. These lesions do not present *in vivo*, sufficiently clear, clean-cut clinical entities. They lack defining signs and symptoms. The symptomatology and other physical phenomena they present are also to be observed in almost all cases of labyrinthitis secondary to otitic suppuration. The clinical and practical classification therefore concerns itself with a consideration of groups of lesions which either require surgery to the labyrinth or which can heal without such surgery. In the latter group are to be found the lesions termed osseous perilabyrinthitis, serofibrinous labyrinthitis, fistula of the lateral semicircular canals or of the promontory, and hematogenous serous labyrinthitis. The latter lesion is the one usually occurring in scarlet fever.

*Perilabyrinthitis.*—By this term is meant a suppurative involvement of the perilabyrinthine cell structures. More often than not, this lesion precedes the complete development of an empyema of the petrous apex. Its development follows in the course of an acute otitic suppuration, and it manifests itself either prior to, or subsequent to surgery upon the mastoid process. There occur transient mild attacks of dizziness which cannot be classified as real vertigo. These mild attacks are often accompanied by vomiting. The attacks are of short duration, and during the interval between the attacks the patient presents nothing of significance. Often the vomiting is

ascribed to dietary indiscretions, or car sickness, etc. The series of attacks last perhaps no longer than five days, and the intensity of the attacks vary from an occasional mild spell during the day to none at all, except that the patient is aware of a constant feeling of uneasiness.

In about half of the cases there appears a slight weakening of the facial muscles on the affected side. This symptom appears about the second day and becomes more pronounced in the succeeding days, only to disappear entirely within from five to seven days. The patient is often up and about, except when dizzy. There is rarely more than a low grade temperature range, but if confined to bed the patient is never obliged to take the "forced position" as in real labyrinthine lesions. When examined functionally all the tests usually employed give either normal reactions or slightly exaggerated responses to stimuli. Either during the course of these series of transient symptoms or at the period of their termination there presents deep retro-orbital pain. There may or may not be diplopia. If headache is complained of, and a lumbar puncture is done, the spinal fluid is found normal in all its essential elements—chemically, cytologically and bacteriologically.

The diagnosis of this condition rests on the comprehension of the transient symptoms, the negative labyrinthine tests and the negative spinal fluid findings and upon the symptoms from the first branch of the fifth nerve. Roentgenograms are essentially negative, although sometimes a haziness of the petrosal pyramid can be established without decalcification or coalescence of its bony cellular structure.

*Scrofulinous Labyrinthitis.*—This is actually a fibrinous involvement of the perilymphatic space of the labyrinth. The tympanogenic variety of this lesion occurs as the result of dialysis of bacterial toxins which pass through an intact round window. Consequently it may, and it often does, accompany an acute purulent otitis media in its early stages. Often it presents symptoms before signs of mastoid suppuration appear. The fibrinous exudate in the perilymphatic spaces in the immediate neighborhood of the round window results in an increase in the amount of perilymph. This has, as a consequence, increased pressure generally in the endolymphatic spaces. Hence violent labyrinthine reactions make their appearance. There is vertigo, vomiting, violent nystagmus which hold to no fixed direction, but may be in all directions. There is, however, a general tendency noted in the nystagmus to take direction away from the involved side. These symptoms come on at once, at the very com-



mencement of the lesion. The patient is bedridden and lies in the typical labyrinthine "forced position." Temperature varies; sometimes it is high, and occasionally low in range. The temperature range is proportionate to the acuteness and the intensity of the underlying otitic suppuration. The lesion itself, confined in a hard bony capsule, preventing systemic absorption, gives no fever. The severe onset gradually subsides after an interval of from four to five days, and the subsidence is enhanced by the removal of the irritating factors in the middle ear.

The use of functional labyrinthine tests (caloric or rotation) are not of much value during the early acute stage because the labyrinthine soft tissues are already in a state of hyperirritability. The reactions which are obtainable by artificial stimulation are already spontaneously present. In cases which present a nystagmus constantly in one direction toward one or the other side, it is possible by either hot or cold caloric tests to get confirmative findings either to stop or reverse the reactions spontaneously presented by the patient. In the early acute stage of the lesion the most important diagnostic test available is the determination of the lateralization of sound toward or away from the affected ear. By the Weber test, it is possible to estimate whether the labyrinthine end organ is alive or destroyed. In certain cases of serofibrinous labyrinthitis, however, a hydrops labyrinthi occurs in which the endolymphatic spaces are so involved in the increased serous transudate that an ectasia of the ductus cochlearis results, the degree depending upon the amount of transudation present. The pressure which results upon Corti's organ produces a definite loss of hearing acuity. Whether this loss in hearing is to be temporary or permanent depends upon how long the excessive pressure is exerted and the degree of destruction it causes to the membranous labyrinthine tissues. Consequently in most cases of serofibrinous labyrinthitis sound will be lateralized toward the opposite ear. In general it may be stated that it is almost impossible definitely to establish an absolute diagnosis and clinically classify a case as one of serofibrinous labyrinthitis. The diagnosis can be made only tentatively. Additional diagnostic aids are to be sought from a study of the arachnoidal reactions as noted in an examination of the cerebrospinal fluid. Aside from its inherent therapeutic value, as one of the means to reduce intracranial pressure generally, and thus beneficently influence a reduction of intralabyrinthine pressure, the fluid may show a slight increase in pressure. Otherwise it is normal. The normalcy of the fluid is a factor toward the establishment of the diagnosis.

*Hematogenous Serofibrinous Labyrinthitis.*—This type of lesion is found in generalized toxemias, and is most often observed accompanying either scarlet fever or mumps. The cochlear portion of the labyrinth is most often affected; the semicircular canals often remain uninvolved. Hence there are few, if any, symptoms of disturbed canal function to be found. The lesion usually occurs in the course of an acute otitis media purulenta during scarlet fever. There is presented a sudden loss of hearing. The Weber test gives sound lateralization to the unaffected ear. The caloric labyrinthine tests give normal reactions. If the spinal fluid is taken, it will give generally negative findings, except as regards lactic acid, which will be increased, but its normal ratio to the lactic acid in the blood plasma will be maintained. This type of lesion is in reality not secondary to the accompanying purulent otitis, but is the result of a serofibrinous exudate and labyrinthine hydrops secondary to the scarlet fever itself.

*Fistula of the Labyrinthine Capsule.*—This lesion is always the result of a middle ear lesion which is capable of causing bone resorption. It is found most frequently to occur in the presence of cholesteatoma, and the horizontal semicircular canal is most frequently involved. The lesion may also result from an acute necrotic otitis (Wittmaack), since this too is a lesion associated with the rarefaction of bone. Such an acute otitis usually runs a long course and is loosely termed "chronic otitis."

The patient begins to evidence transient attacks of dizziness which become more frequent and more intense as time goes on. Labyrinthine tests give exaggerated responses both to the caloric and the rotation tests. If the fistula is located in one of the semicircular canals the specific fistula test gives a positive reaction. If the fistula is located on the promontory the fistula test may be negative. Cholesteatoma most often involves the canal; necrotic otitis less often involves the canal than the promontory, but this rule is not constant.

The data upon which a diagnosis is made consists in obtaining a history of a long standing, persistent otorrhea with a middle ear lesion of the dangerous type, and whether the fistula test is positive or not; this finding in the presence of dizziness, normal labyrinthine reactions, normal equilibratory cerebellar function, makes possible the diagnosis. When the lesion has progressed through the bony fistula into the labyrinthine channels and destroyed them, other types of lesions present themselves. The destruction of the membranous structure places the case in another category.

*Acute Generalized Purulent Labyrinthitis.*—All the types of labyrinthine lesion described above may be followed in the course of

their subsequent development by a lesion which is termed acute generalized purulent labyrinthitis.

The patient presents, at first, a violent nystagmus, directed toward the affected side, having frequent spells of vomiting and vertigo. The patient lies in bed in the "forced position" and generally presents high fever. When tested for lateralization, the patient lateralizes toward the healthy ear. The labyrinth fails to respond to all types of labyrinthine stimulation. Hearing acuity is lost. In the course of time, varying individually, there is a general abatement in the severity of the symptoms. The nystagmus ceases. The patient becomes relaxed, and is able to change position in bed without an attack of vertigo or vomiting. The fever remains high, however, and symptoms of headaches, at first at the occiput and then more generalized, supervene. One of two courses now present themselves. Either the case resolves, and the patient recovers from the labyrinthine involvement with a permanently damaged internal ear, or the patient rapidly progresses toward a meningitis. At first this meningeal lesion is to be considered as protective in nature, and localized in type. Hence during the course of the active labyrinthine reactions to the bacterial invasion it is of great importance to study repeatedly the findings which the spinal fluid will show.

The exact findings in the spinal fluid will vary in the individual case due to the amount of arachnoidal reaction which the patient presents. In general, the pressure will be increased. The copper reduction will often be present, but if examined quantitatively it will be found less than the normal range. In all patients with fever there is an increase in the lactic acid content of the cerebrospinal fluid, when estimations are undertaken, establishing this increase in the fluid to the increase in the patient's blood plasma, the ratio of lactic acid in the spinal fluid will be found higher than the normal ratio between fluid and blood plasma. The spinal fluid chloride, carbonates and pH. will be found showing a tendency to drop below the normal gradients established for normal cerebrospinal fluid in relation to blood plasma. The arachnoid reactions are further evidenced by an increase in cells, globulin and albumin.

The only lesion of tympanomastoid origin from which differentiation is necessary is an abscess in the cerebellum. Cerebellar abscess gives postoccipital headaches, gives nystagmus, vertigo and vomiting; and may also present a patient lying in "forced position." The forced position, however, is generally absent. The Weber gives lateralization toward the diseased ears in cerebellar normal abscess. Hearing is not lost. Caloric tests to the labyrinth evoke normal

responses. The nystagmus starts more gradually and increases in intensity and persists indefinitely until the lesion is terminated by surgery or death. The cerebrospinal fluid gives increased pressure. The copper reduction is at first present; when meningitis develops it generally gradually disappears, and the other findings in the fluid are dependent upon the degree and intensity of the reactions from the meninges. Thus lactic acid is increased in its ratio to blood plasma, and as the meningitic involvement progresses, polynuclear cells appear in increasing numbers in the fluid. The pH., the carbonates and the chlorides are reduced in their gradients between the cerebrospinal fluid and the findings in the blood plasma. Globulin and albumin are present. When distal signs of disorientation, ataxias and other specific cerebellar signs are obtained, the diagnosis of a lesion in the cerebellum from one in the labyrinth is clarified and established.

Finally, a local collection of purulency—a localized meningitis in the pontine angle cistern may simulate symptoms from a purulent labyrinthitis. The presence of nystagmus is variable. It does not present itself as a constant symptom at the onset of the lesion. The direction of the nystagmus varies. Vertigo is inconstant, although vomiting may be present. Hearing is not lost, and the labyrinth will react to artificial stimulation. Weber test shows lateralization to the sick ear. The patient is apt to lie in any position, but mostly on the back.

The cerebrospinal fluid gives the evidence of meningitis. The cytology shows increased polynuclear cells; the copper reduction is either entirely absent or much reduced in quantity. In the early stages and where the lesion is strictly limited, the copper reduction may be normal. Lactic acid is much increased in ratio over the finding of the acid in blood plasma. The gradient between blood plasma and cerebrospinal fluid shows a lowered pH., lowered carbonates and lowered chlorides. There are marked increases of globulin and albumin, and often there are bacteria to be found in the fluid, although in the early stages, where differential diagnosis comes into question, they are usually absent. Later, if the meningeal infection spreads, the clinical picture of meningitis supervenes, and at this time bacteria can usually be found in the fluid.

In the accompanying tables these characteristic findings in each of the lesions described are placed in juxtaposition for ready working reference. The charts do not embrace all the differentiations in detail but constitute a gross working basis for differential diagnosis of peri and labyrinthine lesions.

51 WEST 73RD STREET.

CHART I.—NYSTAGMUS.

	Nystagmus	Duration	Direction
Perilabyrinth	Absent	—	—
Serofibrinous Labyrinth	Present	About 5 days	Toward good ear
Hematogenous Serofibrinous Labyrinthitis	Absent	—	—
Labyrinthine Fistula	Absent	—	—
Purulent Labyrinth	Present	From a few days to several weeks	Toward sick ear
Cerebellar Abscess	Present	Persistent	In all directions
Local Meningitis	Absent or Present	Varies	Varies

CHART II.—DIFFERENTIAL DATA.

	Vertigo	Vomiting	Cochlear Function	Weber	Caloric Test	Fistula Test	Forced Position
Perilabyrinth	Transient	Transient	Normal	Bad ear	Normal	Neg.	No
Serofibrinous Labyrinthitis	Marked	Marked	Impaired	Good ear hydriops otherwise bad ear	Equivocal	Neg.	Yes
Hematogenous Serofibrinous Labyrinthitis	Absent	Absent	Absent	Good ear	Normal	Neg.	No
Labyrinthine Fistula	Transient	Absent	Normal	Bad ear	Normal	Positive	No
Purulent Labyrinthitis	Marked	Marked	Absent	Good ear	Absent	Neg.	Yes
Cerebellar Abscess	Constant but not violent	Occasional	Normal	Bad ear	Impaired due to Central lesion	Neg.	No
Local Meningitis	Absent or present	Present	Normal	Bad ear	Normal	Neg.	No

CHART IIIA.—1 ROUTINE.

Lesion	GROSS DIFFERENTIAL DATA IN SPINAL FLUID.				
	Pressure	Cytology	Bacteriology	Consistency	Albumin
Perilabyrinthitis	Normal	Normal	None	Normal	Absent
Serofibrinous Labyrinthitis	Normal plus	Slightly increased	None	Normal	Absent
Hematogenous Serofibrinous Labyrinthitis	Normal	Normal	None	Normal	Absent
Labyrinthine Fistula	Increased	Increased	None	Normal	Absent
Purulent Labyrinthitis	Increased	Marked	None—later present	Denser than normal	Trace
Cerebellar Abscess	Increased violent	increase	Present or absent	Denser than normal	Present
Local Meningitis	Increased	Marked increase	Present	Denser than normal	More than present
					Markedly present

CHART IIIB.—CHEMICAL.

Lesion	GROSS DIFFERENTIAL DATA IN SPINAL FLUID.				
	Lactic Acid	Chlorides	Copper Reduction	Carbonates	Ph.
Perilabyrinthitis	Normal Ratio to B. P.	Normal	Present	Normal	Normal
Serofibrinous Labyrinthitis	Normal Ratio to B. P.	Normal	Present	Normal	Normal
Hematogenous Serofibrinous Labyrinthitis	Normal Ratio to B. P.	Normal	Present	Normal	Normal
Labyrinthine Fistula	Normal Ratio to B. P.	Normal	Present	Normal	Normal
Purulent Labyrinthitis	C. S. F. Ratio to B. P. increased	Slightly reduced	Present but less in amount than normal	Trend toward reduction	Lowered slightly
Cerebellar Abscess	C. S. F. Ratio to B. P. increased	Reduced	Present or absent	Less than normal	Lowered
Local Meningitis	Marked increase of C. S. F. to B. P.	Reduced	Absent or present in lessened amount	Distinctly lowered	Lowered

LIX.

OPERATION FOR REMOVAL OF CARCINOMA OF THE  
FAUCIAL TONSIL AND CONTIGUOUS PARTS.\*

DUNCAN MACPHERSON, M. D.,  
NEW YORK.

The difficulty of reaching the base of the tongue, the lower pole of the tonsil and the lower parts of an involved oropharynx when invaded by a malignant neoplasm led me and my associate, Dr. W. Alex. Newlands, to work out on the cadaver and apply the following method in operating on two patients, on one of which we operated together and one which he operated on. The external carotid artery is tied as far from the bifurcation as its branches will permit, with heavy silk and slow stricture. Rapid tying or a thin ligature ruptures the intima endangering the patient to aneurysm of the middle and external coats. The ascending pharyngeal artery, especially if coming from the bifurcation, may need tying at the same time. Lymphatic glands may be removed at this time. It has been a surprise to me, however, to find so many patients free from palpable or macroscopic glands with the pharynx involved by malignancy.

The second stage is carried out in about two weeks when an incision is made from the symphysis of the inferior maxilla to the suprasternal notch, the lower extension being made lest the insertion of a tracheal tube should be considered necessary. The skin, superficial fascia and platysma of the affected side are reflected back without making lateral incisions. The corresponding half of the hyoid bone is uncovered and removed, leaving the periosteum attached to the uncut fibers of the muscles. The digastric attachment to the hyoid is separated and lifted upwards, as is the submaxillary gland. The mylohyoid muscle is separated from the attachment to its fellow of the opposite side in the median line and it, together with the geniohyoid, stylohyoid, middle constrictor, geniohyoglossus, mucous membrane, and possibly the hyoidglossus, are severed laterally for a sufficient distance to permit of easy access and a good view of the lower pole of the tonsil and the surrounding area. Important structures brought into view and to be preserved during this phase of the opera-

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\*Read before the annual meeting of the American Laryngological Association, at Cleveland, June 8, 1934.

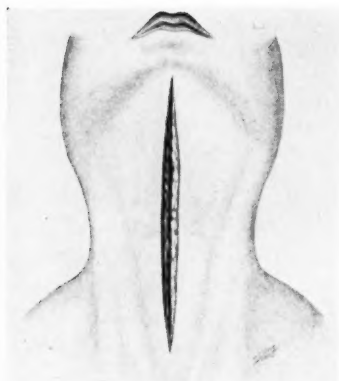


Fig. 1.

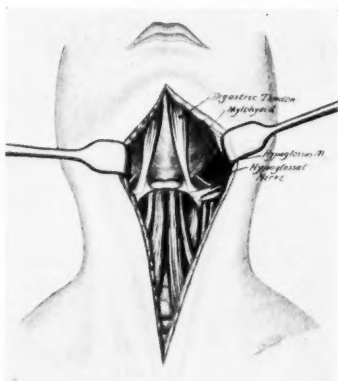


Fig. 2.

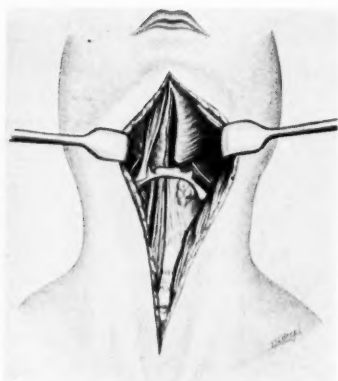


Fig. 3.

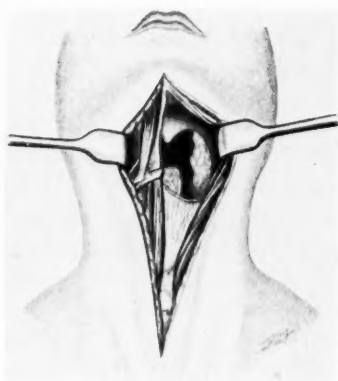


Fig. 4.

Fig. 1. Showing primary incision extended to suprasternal notch lest for any reason tracheotomy might be necessary.

Fig. 2. Showing digastric tendon, hypoglossal nerve, hyoid bone, mylohyoid and hypoglossus muscles.

Fig. 3. Mylohyoid, genioid and mucous membrane cut in median line and the hyoid bone loosened from attachments. The hyoid may now be cut in median line and enucleated from its bed—leaving the uncut fibers of its upper and lower muscular attachments in situ.

Fig. 4. Showing the hyoid cut and removed, the diseased tonsil and epiglottis. The superior muscular attachments of the hyoid are cut laterally and drawn aside together with the hypoglossal nerve and submaxillary gland.



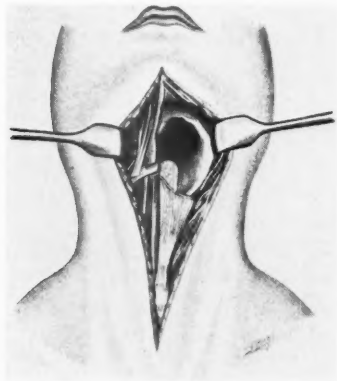


Fig. 5.

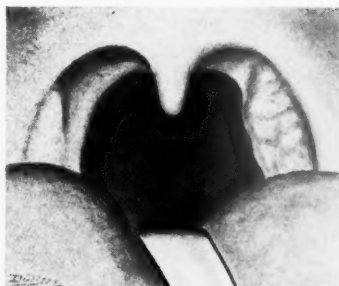


Fig. 6.

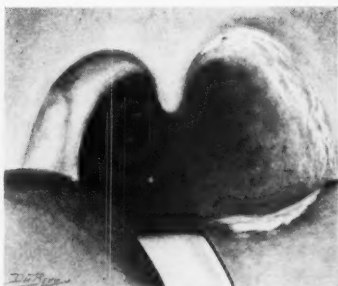


Fig. 7.

Fig. 5. Shows diagrammatically the diseased area after removal of the growth as seen from the pharyngeal opening.

Fig. 6. Shows the tonsil and tongue as seen through the mouth.

Fig. 7. Shows the lateral wall of the pharynx, base of the tongue and part of the soft palate as seen through the mouth after removal of the growth.

tion are the hypoglossal, perhaps the glossopharyngeal and superior laryngeal nerves. Before operating the entire anatomy should be reviewed on the cadaver.

Diathermy was used on the cadaver and on the living. The cutting current was used almost exclusively, but the coagulating current was used to control the very slight hemorrhage—although the entire operation is almost devoid of bleeding. The upper pole of the tonsil, the involved palate and part of the base of the tongue may be reached and operated upon through the mouth. The external surface of the tonsils lies on the superior constrictors which separate them

from the internal carotid arteries, internal jugular veins, pneumogastric nerves, etc., while the middle constrictors are in relation with the carotid vessels, the existence of which relationship must be borne in mind.

A feeding tube is placed in position through the nose and feeding is carried out for a longer or shorter period of time, depending on the convalescence and circumstances attending each individual case. The hyoid muscles, upper and lower, are brought together and sutured, the external structures sutured and a drain placed in the lower angle of the wound. When healing has taken place we have found in these cases and in cases of laryngectomy with central pharyngotomy, in which latter cases we removed the hyoid completely in order to reach a diseased pharynx—that deglutition and other functions of the hyoid muscles were not interfered with. The two stage operation was resorted to, believing that thereby the neck structures would be protected by new cicatricial tissue from dirty mouth infections. The operation is so easily done that it may be performed by anyone who can tie the external carotid.

One thinks of several modifications of this operation. For instance:

1. Radium may be used before and after removal of the diseased tissue.
  2. The entire operation might be done at one time, an advantage being that for greater safety the internal carotid could be under control either by a ligature placed under it or under the common during the operation.
  3. The lingual, facial and ascending pharyngeal arteries might alone be tied, leaving the external carotid itself patent.
  4. The wound might be left open, as I have done in central pharyngotomies when accompanying laryngectomy—in which cases, realizing that breaking down and sloughing of the pharynx would be inevitable, we felt that the parts would be more easily reached by bacteriophage or other local applications, suction, etc., from the outside. The parts granulate quite rapidly in such cases, manipulation and dressings being carried out under direct vision.
  5. The parts might be approached by a lateral pharyngotomy, but the exposure did not seem to us as easy or as open as the central.
- The merits or demerits of radium treatment used alone in parallel cases is not under consideration in this paper.

140 EAST 54TH STREET.

## LX.

### HEREDITARY HEMORRHAGIC TELANGIECTASIA.\*

KARL MUSSER HOUSER, M. D.,

PHILADELPHIA.

Since Osler,<sup>1</sup> in 1901, published his paper on "A Family Form of Recurring Epistaxis Associated with Multiple Telangiectases of the Skin and Mucous Membranes," much has been written concerning this condition, and a number of other families subject to this disease have been reported. In 1896, however, Rendu<sup>2</sup> had called attention to the association of multiple telangiectases with familial epistaxis, hence the condition is sometimes called Rendu-Osler's disease or Osler's disease. In the past few years the names of other authors have been associated with this malady, but for the sake of simplicity of nomenclature it seems best to refer to it by the adequately descriptive but sufficiently complex title of "multiple congenital telangiectasia," without still further confusing the subject by the addition of the name of each author who chooses to record his experiences.

This disease is well known to the internist and hematologist, but should be brought to the attention of the rhinologist, because epistaxis is frequently the initial symptom. Most of the literature on this subject has appeared in journals other than those devoted to rhinology, and unless the collateral reading of the rhinologist has been rather widespread, or he has had contact with the disease, he has little or no knowledge of its peculiarities.

The latest authoritative, comprehensive review of this subject, by Hurst and Plummer,<sup>3</sup> in January, 1932, reported that the literature up to that time included but fifty-seven family trees, which showed indisputable evidence of the disease in its true form. Goldstein<sup>4</sup> has reported a larger number, but exception may be taken to some of the families and cases included. Usually not more than two or three cases were observed from each family tree, so that it is safe to assume that the total number of patients actually treated and observed is less than 200, although the total number affected and

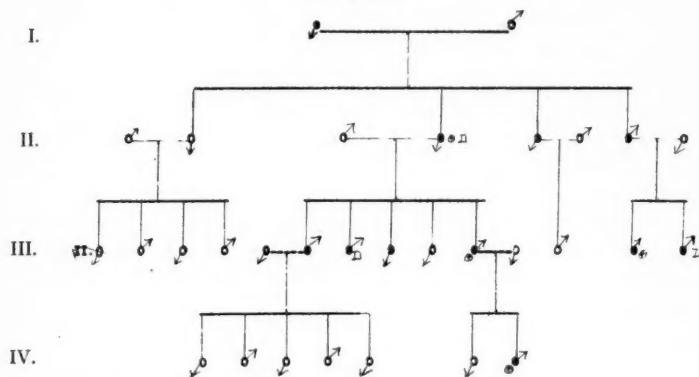
\*Presented before the annual meeting of the American Laryngological Association at Cleveland, June 8, 1934.

From the Department of Otolaryngology of the University of Pennsylvania, Philadelphia, Pa.

included in the family trees would probably reach eight or nine hundred. One need not quibble, however, about the exact number of authentic cases occurring to date, it being far more important to become familiar with the disease itself, so that its recognition will not escape us, and that we become more proficient in the treatment of those unfortunate enough to be afflicted.

Chart No. I, illustrating a typical family tree in which this disorder is present, will serve as an introduction to this condition. Here a history of epistaxis of varying degree runs through four gen-

CHART I.



Epistaxis with hereditary hemorrhagic telangiectasis:

- |   |                            |              |
|---|----------------------------|--------------|
| ♂ | Males                      | } Unaffected |
| ♀ | Females                    |              |
| ♂ | Males                      | } Affected   |
| ♀ | Females                    |              |
| ⊕ | Studied by the author      |              |
| D | Dead from nasal hemorrhage |              |

erations. Two of these patients have been mentioned previously by Fitz-Hugh,<sup>3</sup> but the family as a unit has never before been reported. The author has had the opportunity of studying and treating four members of this family, one from the second, two from the third and one from the fourth generations. Of these four patients one is dead from nasal hemorrhage, two other members of the family having died from the same cause.

Hemophilia is the disease with which multiple hemorrhagic telangiectasis is most likely to be confused. A careful history, however, usually will exclude the former disease.

Hemophilia does not often, if ever, affect females. Stevens<sup>6</sup> says:

"Males of bleeder families are prone to inherit the disease, but virtually never transmit it to their descendants unless they themselves are bleeders. Even males who are themselves affected are much less likely to transmit the tendency than healthy females of hemophilic parentage. Although daughters of a bleeder, even if healthy, commonly transmit the disease to their male offspring, they sometimes fail to do so. The second generation is usually skipped, and occasionally the descent is interrupted for two generations."

The essential blood changes of hemophilia are a prolongation of the coagulation time, probably due to some chemical deficiency not definitely known, as the amount of platelets, fibrin and blood salts are normal. In addition, bleeding time and blood clot retraction show no evidence of abnormality.

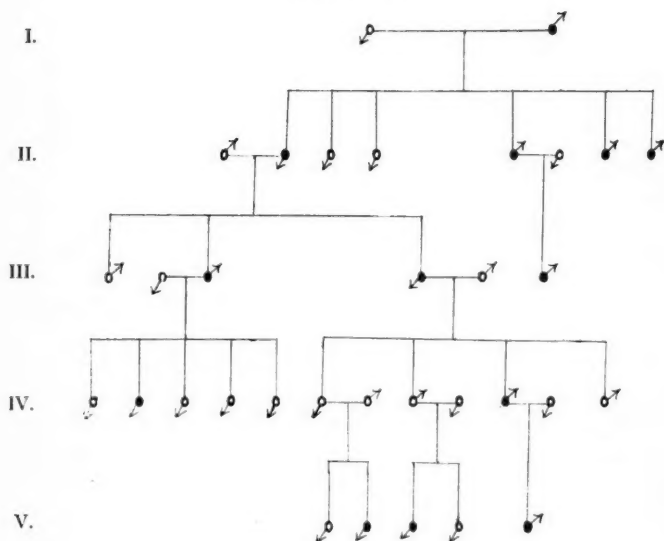
In multiple hemorrhagic telangiectasia males and females may both be affected and transmit the disease. Fitz-Hugh<sup>7</sup> has called attention to atavism, which sometimes occurs and is illustrated by Chart No. II, which shows an example of atavism in a family tree reported by Edel,<sup>8</sup> a skipped generation occurring between the third and fifth generations in one branch of the family. The blood changes here are none other than those dependent upon the degree of secondary anemia present. There is no tendency to bleed readily from cuts, the hemorrhage always occurring from one or more of the naevi, which must be present to allow the case to be properly classified as one of multiple hemorrhagic telangiectasia.

According to Larrabee and Littman,<sup>9</sup> three postulates must be fulfilled before the diagnosis can be made:

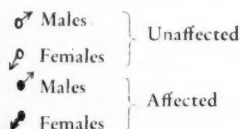
1. There must be a history of heredity.
2. There must be visible telangiectases in numbers and distribution distinctly pathologic.
3. There must be a tendency to bleed from such lesions.

*Lesions.*—The lesions in telangiectasia are quite similar to those frequently seen in apparently normal individuals. Usually they are found as small red or purple spots, superficially similar to those of purpura, on the nasal mucosa, where they may be present on both the septum and turbinates. A few, and sometimes many, can frequently be found on the oral mucosa. They have been found on the inner side of the lips and cheeks, gums, tongue, palate and larynx (Kofler<sup>10</sup>). They have been discovered in the stomach and intestines, urinary tract, brain and liver. The external distribution may vary, and many, few or none may be found. Here they have been described on the face, ears, conjunctiva, trunk, and frequently on the finger tips. They may be distinguished from purpura by pressure.

CHART II.



Family reported by Edel<sup>8</sup>, showing atavism in the fifth generation:



If a purpuric spot is subjected to pressure it does not disappear, whereas the opposite is true of a naevus. Bleeding may occur from any of the lesions, but is by far most common from those of the nasal mucosa, those of the oral mucosa being next in order of frequency. In fact, it is epistaxis that calls attention to the disease in the vast majority of cases. Various writers have reported cases where bleeding occurred from the finger tips,<sup>11</sup> scalp, ears,<sup>12</sup> and urinary tract.<sup>13</sup> The bleeding is apparently entirely dependent upon the presence of the naevi, the blood elements, such as calcium, platelets, bleeding time, clotting time and clot retraction being normal.

The microscopic appearance of these naevi has been reported by Hones,<sup>14</sup> Steiner<sup>15</sup> and Arrak,<sup>16</sup> each of which comment upon the extreme thinness of the vessel walls. These walls are nothing more than a single layer of endothelial cells covered by a greatly thinned epithelium, which is sufficient to account for their marked tendency

to hemorrhage. Ullman<sup>17</sup> states that the connective tissue of these vessel walls undergo rarefaction changes of the connective tissue similar to parenchymatous degeneration.

Two explanations of hemorrhage in these cases have been offered. One calls for blood dyscrasia of undetermined nature; the other, that hemorrhage is simply the result of a rupture of the above mentioned vascular structures.

*Prognosis.*—Death from epistaxis in this condition is relatively rare, there being a mortality of approximately 4 per cent<sup>7</sup> due to the disease itself in the families previously reported. In the family mentioned here, there have been three deaths in eleven cases, a death rate of 27.2 per cent. Even though the danger of death from this type of bleeding is relatively slight in most cases, the hemorrhages tend to increase in severity and frequency toward middle age, and if not checked lead to moderate or marked secondary anemia, with the attendant phenomena and perils of this weakened condition. Hence many, although not dying of the disease itself, are cut down before their time by conditions induced by the constant loss of blood.

*Treatment.*—In three of the author's cases simple chemical cauterizations, i. e., chromic acid, of the lesions as they appeared has been rather effectual. This rather supports the mechanistic causation theory, in that if the naevi are destroyed the bleeding ceases. In one case, however, no matter how thoroughly the lesions were destroyed, new ones formed in the adjacent mucosa in a relatively short period of time. The new naevi showed just as much tendency to bleed as the initial lesions. It was this case in which fatal hemorrhage occurred about a year after treatment was discontinued. Some authors have reported good results from radium.<sup>18 19</sup> Neither radium, X-ray, electrocoagulation nor galvanocautery have given as good results, in the hands of the author, as the chromic acid bead. Trichloroacetic acid does not seem to form enough scar tissue to give permanent results. Sometimes a septal perforation will follow the repeated use of chromic acid, but this is much to be preferred to the constant bleeding. General measures, such as high vitamin anti-anemic diets, serum therapy, organotherapy and cobra and rattlesnake venom antiviruses, have been used and advocated by some writers. With the exception of those agents which tend to combat the existing anemia and build up general bodily resistance, the author has had no experience.

Hurst and Plummer<sup>3</sup> have used a simple device by which the patient himself can control the hemorrhage. He carries with him a soft rubber catheter which passes into a rubber finger stall as far as it will go. The neck of the stall is tied tightly around the catheter,

but not tightly enough to occlude its lumen. This may be smeared with vaselin or dipped into oil and forced by the patient along the floor of the nose as far as the neck of the stall. The rubber bag is then inflated and pressure maintained for ten or fifteen minutes. Deflation is slowly performed and the bag allowed to remain in place for a few minutes, either to drop out or be gently extracted. If both sides are bleeding, they are inflated at the same time. This procedure, which is a modification of the Cooper-Rose inflation plug, can be repeated as often as necessary.

*Transfusion.*—In some of these cases such profound bleeding occurs that transfusion may seem necessary. Fitz-Hugh,<sup>5</sup> however, calls attention to the increasing intolerance to blood transfusions in this disease, especially where there is splenomegalia and hepatic enlargement in the individual who falls in the type IV (Moss) blood group. Four such cases are reported by him, two of which died following blood transfusion. Therefore, it is wise to consider the case carefully before resorting to transfusions.

#### REPORT OF CASES.

CASE 1.—Mr. E. H. S. The first of these cases seen by the author was a 35 year old male, who came to his office in March, 1929. His history was typical in that it was an hereditary trait. Epistaxis was profuse but relatively infrequent, usually occurring not more often than once in six or eight weeks. At this time he would have bleeding attacks for several days, and then have relatively little difficulty until his next major hemorrhage. Several naevi were found upon the anterior inferior portion of the nasal septum, but none elsewhere. Suspecting some blood disorder, he was referred for blood studies to Dr. Thomas Fitz-Hugh, Jr., who made a positive diagnosis of hereditary hemorrhagic telangiectasia. Nothing was done with this patient, other than chromic acid cauterization of the nasal lesion, which after a number of treatments resulted in a complete absence of hemorrhage for the past three and one-half years.

CASE 2.—Mrs. J. S. S., the mother of No. 1, who when first seen was 58 years old. She had suffered from repeated nasal hemorrhages since childhood. Treatment of many kinds had been used which included radium, X-ray, electro-coagulation, thermal cautery and blood transfusion. Her nasal interior showed many naevi on both sides, located on the septum and turbinates in the anterior portion. These lesions bled profusely upon the slightest manipulation; in fact, on one occasion such profuse bleeding occurred during the office examination that it was necessary to transfer her to the hospital before the hemorrhage could be controlled.

She, too, was referred to Dr. Fitz-Hugh, who reported a profound secondary anemia with red blood cells 3,000,000, hemoglobin 34 per cent, and the white count normal, other than slight leukopenia. The blood platelets were normal, but there was marked poikilocytosis and anisocytosis with slight polychromatophilia. Blood Wassermann, coagulation time and bleeding time were normal.

In addition to the nasal naevi, numerous telangiectases were present on the labial and nasal mucosa, as well as the skin of the face, hands and neck. The spleen and



liver were both enlarged and palpable. She had received intensive treatment at an earlier date in New York City, and had reacted badly to blood transfusion, having developed jaundice following this procedure. Her general treatment consisted of an anti-anemic diet and deep X-ray therapy of the spleen. Locally, chromic acid cauterization was used, and for a time seemed to control the condition. Later, however, new lesions developed adjacent to the treated areas and even in the treated areas themselves. She eventually became discouraged and failed to return for observation and treatment. From another member of the family it was learned that she died from nasal hemorrhage about a year ago.

CASE 3.—Master H. A. S., the grandchild of Case No. 2 and son of Case No. 1, was seen in March, 1929. This child, age 6 years, had occasional nasal hemorrhage, and examination showed several naevi on the nasal septum. There was no history of undue bleeding from other causes, such as cuts or abrasions. Several cauterizations with the chromic acid bead have completely controlled the epistaxis up to the present time.

CASE 4.—Mr. W. P. In August, 1933, this patient was seen in consultation with the medical service in the Wards of the University Hospital. His history was typical of multiple hereditary telangiectasia, and on questioning him it was discovered that he came from the same town as the three previously mentioned cases, and he was a cousin of Case 2 and an uncle of Case 3. There was no difficulty concerning his diagnosis when the family history was learned and blood studies were completed. He showed the typical nasal naevi of the septum, and several on the anterior ends of the middle turbinates. A few naevi were present on the ventral surface of the tongue. His bleeding dated from early childhood, and at times was so severe as to almost lead to exsanguination. On admission he showed a profound secondary anemia, emaciation, weakness, pallor and splenomegalia. His hemoglobin was 32 per cent, red blood cells 2,500,000, with the other red cell changes common to this degree of blood depletion. On account of the unfavorable reactions reported following transfusions in this type of case, transfusion was not given. Instead a high vitamin, anti-anemic diet with iron by mouth and liver extract intravenously were administered. Local nasal treatment consisted of nothing other than the chromic acid cauterization of the lesion. This immediately brought the nasal bleeding under control, and finally resulted in complete stoppage of the epistaxis. In a month he was discharged from the hospital with a blood count of 4,300,000 red cells and hemoglobin of 65 per cent.

Since that time he has reported once each month. So far there has been no hemorrhage other than an occasional blood streak, when a scab or crust is dislodged from the area that was subjected to cauterization, a septal perforation having occurred in this region.

#### SUMMARY.

1. A family tree showing definite evidence of multiple hemorrhagic telangiectasia is reported.
2. Eleven members of this family in four generations have been afflicted.
3. A mortality of 27.2 per cent has occurred from nasal hemorrhage in these eleven cases.
4. Four members of this family representing three generations have been observed and treated by the author.

5. Chemical cauterization of the lesions has proved of greater value than other forms of local treatment.

6. Attention is called to the danger of transfusion in these cases where hepatic enlargement and splenomegaly exist.

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LXI.

JACOBSON'S ORGAN (ORGANON VOMERO-NASALE,  
JACOBSONI): ITS ANATOMY, GROSS, MICRO-  
SCOPIC AND COMPARATIVE, WITH SOME  
OBSERVATIONS AS WELL ON ITS  
FUNCTION.\*

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In 1811, Ludwig Levin Jacobson (B. Copenhagen, 1783—D. 1843), a Danish military physician and anatomist, described a peculiar diverticulum observed by him in the nasal cavity of certain mammals, i. e., ruminants and rodents. This diverticulum and its accessory structures, situated in the medioventral part of the nasal chamber and now known as Jacobson's organ, was associated by him with Stenson's duct (canalis incisivus Stenonis) and considered by him to be a glandular accessory olfactory organ.

Jacobson, however, was not the first to observe the organ in question. The canal and its nasal orifice had been observed in man and described by Ruysch, in 1703, who, however, attached no particular significance to it. Sömmerring also figured it in his work on the organ of smell, published in 1809 at Frankfurt.

A fairly extensive literature has grown up around the subject of Jacobson's organ, as it has been found in fishes, amphibians, reptiles and in various mammals, including man, in varying stages of development. In fishes it is rudimentary and in the human species Jacobson's organ is retrogressive; it is, however, almost constantly found in the human embryo and with diligence may occasionally be found in the adult.

As most of the important literature concerning Jacobson's organ is to be found in the foreign anatomic, zöologic and other specialized journals, and as there is practically no mention of it in American rhinologic literature or textbooks, a summary of what is known con-

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\*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, 1934.

From Ear, Nose and Throat Department of the Michael Reese Hospital.

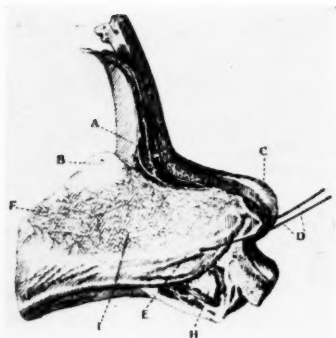


Fig. 1.



Fig. 2.

Fig. 1. This illustration from Ruysch (1703) is the first one to show Jacobson's organ. A probe can be seen entering its orifice. Ruysch had no idea of the nature or function of the organ.

Fig. 2. View of adult human septum. A probe is seen in Jacobson's organ. This illustration is from Sömmering (1809) and is the second time the organ was pictured.

cerning it may be of great interest to rhinologists here who have not already made themselves acquainted with it; moreover, there is some reason to believe that in certain cases it may be associated with pathologic conditions arising in the nose. This would give the organ more than an academic interest.

A large part of the literature of this subject consists of special detailed histologic descriptions of the findings in various genera and species, of the peculiarities of Jacobson's organ and of its connections with the true olfactory system and must be presented in summary only so far as may be necessary in dealing with the general subject. It is more particularly desired in this paper, after having treated the subject in general, to discuss these findings mainly as they relate to the supposed functions of Jacobson's organ and to its occurrence in man.

#### HISTORICAL.

Shortly following Jacobson's communication there were a number of other publications, most of them adding very little to the original description (F. J. Meckel, 1820; Reiffstock, 1823; Rosenthal, 1827). Gratiolet (1845) published the first important histologic study. v. Leydig (1857), in his "Lehrbuch der Histologie," also described the histology. He examined cats and young goats and noticed numerous mucous glands about the organ and also that it had two

nerve supplies, namely, from the olfactory and the fifth nerve. He says very little else about the finer structure, stating that it resembles the rest of the nasal passages and has undoubtedly an analogous function.

The occurrence of Jacobson's organ in sheep was reported by Balogh in 1860, by v. Brunn in 1892 and by Raugé in 1893; Klein in 1881 found it in guinea pigs and dogs; Burckhardt (1891), Sarasin, P. and F. (1890); Bawden (1894), Seydel (1895), Anton (1908, 1911), Zuckerkandl (1910), Bruner (1914), as well as others, described the organ in amphibians. Seydel's work was particularly complete. Similar work for the reptiles was that of Howes (1891), Sluiter (1892), Wright (1893), Röse (1892-95), Leydig (1897) and McCotter (1917). Ganin (1890) described the organ in birds. Klein (1881), Herzfeld (1888), Symington (1891), v. Lenhossek (1892), Broom (1895), Nusbaum (1896), Elliot Smith (1895), Profidia (1904), Christie-Linde (1914) and Minett (1925) furnished studies in various mammals; Wiedersheim (1901) and Frets (1912) described it in primates. Studies on the organ in human embryos and adults have been made among others by Dursy (1869), v. Kölliker (1877), Romiti (1884), Piana (1891), Potiquet (1891), and Röse (1893).

v. Mihalkovics, in 1899, published an elaborate report on the subject. This splendid contribution, consisting of nearly one hundred pages of text and seventy-nine plates, covered the literature to this date, and included the author's special studies of the organ in human embryos. The illustrations reproduce examples of histologic sections of the organ in the various classes and orders in which it was then known to exist, including the human embryo in different stages.

Jacobson's, or the vomero-nasal organ, has not been found to occur in any order lower than the fishes, and it is doubtful if a true Jacobson's organ, rather than a homologue of it, occurs in them. In most reptiles and in the higher amphibia, the organ is highly developed in addition to the true olfactory organ; in fact, in some reptiles, such as lizards and snakes, Jacobson's organ is an incomparably finer sense organ than the olfactory organ proper and is clearly differentiated from the true organ of smell.

In birds Jacobson's organ is said to be absent; the olfactory development in general is atrophied in this group. One observer, however, claims to have observed its presence in embryonic life (Ganin, 1890). In various mammals it has been described with a greater or lesser degree of development, the higher development being in the

lower mammalian types. In man and the primates (Frets, 1912-13) the organ occurs only as a retrogressive remnant without any apparent function.

As stated, the order of fishes is the lowest in which Jacobson's organ has been found. Gawrilenko (1910) found possibly its rudiments in them. As Parker (1922) observes, speaking of Gawrilenko's observation, even in the lowly rays and sharks each olfactory sac is divided into two compartments with separate innervations; these two compartments may be supposed to correspond, one to the vomero-nasal and the other to the olfactory organ proper. This duplex compartment Gawrilenko also found in salmon.

Coming to the amphibia, a discussion of Jacobson's organ in these leads one into a complicated and controversial field. Nevertheless, a proper understanding of the respiratory and olfactory mechanism in this group is necessary if one is to have a comprehensive idea of the forms and functions of the vomero-nasal organ as it is seen in the long range of animals in which it appears. For a clear discussion of the amphibia Nemours (1930) recognizes that a classification of the amphibia should be present in the mind of the reader and his use of Parker and Haswell's classification is likewise adopted here.

#### ORDER I. URODELA.

(a) *Perennibranchiata*.—Those forms that retain gills throughout life. *Proteus*, *necturus* and *siren* are examples of this group.

(b) *Derotremata*.—Gills are lost in the adult stage but retain a gill cleft. Examples of this group are *amphiuma* and *cryptobranchus*.

(c) *Myctodera*.—In the adult the gills and gill clefts are lost. Salamander and triton represent this group.

#### ORDER II. ANURA.

This order has no tail, but four legs are present in the adult. Represented by frogs and toads.

#### ORDER III. GYMNOPTIONA.

No legs in this group. Represented by limbless caecilians.

#### ORDER IV.

Represented by the stegocephali, an extinct species, which were probably the precursors of the above mentioned orders.

Bruner, in 1914, published an important article which helps to clear up many of the controversial points and the data in his contribution are very freely drawn upon in the following: Speaking of respiration and smell in amphibia, he states that all typical members of this group, both the terrestrial and aquatic forms, attain a part of their oxygen supply by means of a buccopharyngeal process in which

the respiratory medium is renewed by regular oscillations of the floor of the mouth. The medium, be it air or water, employed in this process is taken through the nasal cavity and is used as the medium of smell. Bruner proceeds to discuss the structure of the olfactory organ in different groups of amphibia; as shown by Wiedersheim (1879), the Sarasin brothers (1887) and Zuckerkandl (1910), *Gymnophiona* have as an olfactory organ a true olfactory chamber and a well developed Jacobson's organ, the latter opening into the former at the choana. While the choana cannot be closed, the anterior nares can be and are by a sphincter. Adult *Gymnophiona* are air smellers.

In the higher urodeles the nasal organ contains a true olfactory cavity and a maxillary cavity containing the organ of Jacobson. The external nares are closed and opened by a smooth muscle mechanism while the choana is a nonclosing cleft whose median part opens into the olfactory chamber and the lateral part, as suggested by Seydel (1895), provides an open way between the oral cavity and Jacobson's organ.

The ordinary medium of smell in these forms is air, which passes through the nares both in pulmonary and buccopharyngeal respiration. The latter is the chief means of renewing the olfactory medium as shown by the fact that degeneration of the lungs is not followed by degeneration of the olfactory organ. Some adult salamanders (*amblystoma*) may, it is true, breathe by water (aquatic respiration), in which case, of course, water passes through the nasal cavity but it is not known whether this aqueous respiratory medium is also used for olfactory purposes.

In frogs (*Anura*), the choanae are similar to those in the higher Urodeles. The external nares are opened and closed by a striated muscle mechanism; the olfactory organ is complicated and consists of a true olfactory chamber, a maxillary cavity and three blind lateral sacs, (a) superior, (b) middle and (c) inferior. The last one contains Jacobson's organ. Through the maxillary cavity Jacobson's organ is easily reached by the outgoing respiratory current (Gaupp, 1904). Air is the usual medium of smell, and water does not enter the nose under ordinary circumstances.

*Cryptobranchus* and *amphiuma* resemble the higher urodeles in the general structure of Jacobson's organ (Anton, 1908). In the *cryptobranchiata* the choana at valvular and under control of striated muscle. They have a smooth muscle mechanism for opening and closing the anterior nares, hence possess two mechanisms for closing the nasal passages. The smooth muscle sphincter operates during



pulmonary respiration. Water is the medium of smell in amphiuma and cryptobranchus.

Siren has a well developed olfactory organ and a large Jacobson's organ; Wilder (1891), Seydel (1895) and others. A closing mechanism is absent for the anterior nares, but a complicated one for the choanæ is present.

In necturus and proteus the nasal cavity is simple, almost tubular, and no Jacobson's organ is present, Seydel (1895), Anton (1911). According to Anton, Jacobson's organ is represented by a lateral row of olfactory buds which is separated from the median part of the nasal cavity by a strip of indifferent respiratory epithelium. The olfactory mechanism in necturus and maybe proteus is respiratory water. This passes through the nasal cavity into the mouth and is expelled through the gill clefts. Respiratory air enters by way of the oral cavity and does not normally enter the nasal cavity.

On the basis of his detailed and exhaustive examination of the respiratory mechanisms and their manner of functioning Bruner (1914) agrees substantially with Seydel that in the higher amphibia the nasal organ consists of two physiologically and anatomically distinct parts, (1) a true olfactory cavity to test respiratory currents, and (2) an organ of Jacobson which receives its stimulation by way of the expiratory current. This state of affairs prevails, no matter whether the olfactory medium is air or water. Bruner summarizes his opinions as follows:

1. In all the amphibia he examined the medium of smell is usually the same as that used in buccopharyngeal (or branchial) respiration. This medium is renewed by regular oscillations of the floor of the mouth.

2. The olfactory medium may be air or water in adult amphibia. The olfactory organ reaches a higher state of development in air smellers than in water smellers. There is, however, no marked difference between the olfactory organ of the higher water smellers (amphiuma and cryptobranchus) and that of the lower air smellers as amblystoma. Jacobson's organ is present in all these forms.

3. Different mechanisms are used to control the olfactory (respiratory) medium in different groups and even in different stages of the same individual. These mechanisms fall into two types:

- (a) The respiratory medium passes freely through the nasal cavity into the mouth, but its return to the nasal cavity from the mouth is stopped by a choanal valve.



(b) The respiratory mechanism in this second type is wholly under muscular control and the olfactory medium passes freely in and out through the nasal cavity.

Corresponding to these two types of respiratory mechanism one can distinguish—

(a) Monosmatic forms (single smellers), including *necturus*, larvæ of *amblystoma* and *rana*, in which the olfactory organ is used to test only the external medium. He proposes the name *Monosmesis* for this condition.

(b) Diosmatic forms (double smellers), including *siren*, *crypto-branchus*, *amphiuma*, the larvæ of lungless salamanders and the adult stage of higher amphibia. In these forms ingoing and outgoing currents bear odorous matter to the olfactory organ which is accordingly used to test both the external medium and the contents of the oral cavity. This condition, which is common to all higher vertebrates, Burner suggests, may be called *diosmesis*.

In adult single smellers (*necturus*) Jacobson's organ is wanting and the olfactory organ is simple. In double smellers the olfactory organ is complex and Jacobson's organ is present. For a complex olfactory organ to develop with the presence of an organ of Jacobson it makes less difference whether the olfactory medium is either air or water than whether the animal is a single or double smeller, i. e., the nature of the olfactory medium is of less importance than the question whether the animal is a single or double smeller.

In the class of reptiles, Jacobson's organ is highly developed in snakes (*ophidia*), and lizards (*lacertilia*). It was first seen in this order by Rathke (1839), who thought it was a gland and so missed its significance. Stannius (1854) first recognized its true nature and described it; later Leydig (1872). Born (1877-79-83), in his studies of the nasal cavities, makes note of it; Mihalkovics (1899) deals with it at great length, and Katheriner (1900) notes its marked development in water snakes, and because of this suggests its importance in these forms as a sensory organ. The organ in snakes is separated from the nasal passages and opens directly into the mouth, but the passageway is quite narrow. In lizards the findings are similar to those in snakes, but the organ does not reach quite so high a degree of development. Mihalkovics (1899) was deeply impressed in lizards and especially in snakes by the important state of Jacobson's organ; the great thickness of the sensory cell wall in snakes, often as much as 200  $\mu$ . thick, and its rich supply of nerves led him to believe that it was a highly differentiated mechanism with an important func-

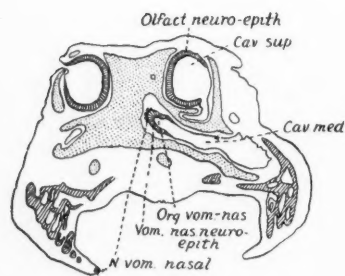


Fig. 3.

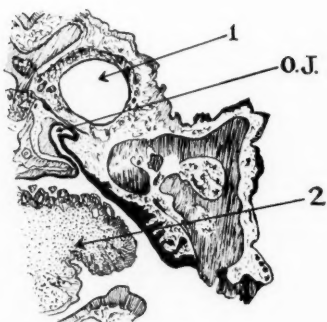


Fig. 4.

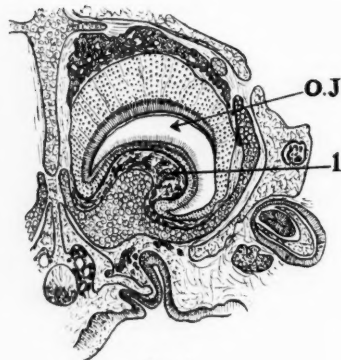


Fig. 5.

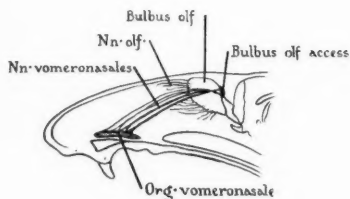


Fig. 6.

Fig. 3. (After McCotter.) A transverse section through the vomero-nasal organ of the frog, showing the relation of the superior and middle nasal cavities and the distribution of the vomero-nasal and olfactory mucosa. McCotter places the vomero-nasal organ in the cavum media and indicates the vomero-nasal nerves.

Fig. 4. (After Nemours.) Section through the nasal cavity (1) and organ of Jacobson (O. J.) of adult chameleon (*anolis carolinensis*). The organ does not communicate with the nasal cavity and opens directly into the mouth.

Fig. 5. (After v. Mihalkovics.) Frontal section through the nose and organ of Jacobson in an adder (*coluber natrix*). The respiratory epithelium is low and ciliated. The lumen of the organ is encroached upon by a projection, (1) the eminentia fungiformis. The sensory epithelium is highly developed, showing the cells in peculiar strand-like formations.

Fig. 6. (After McCotter.) A median section of the head of a rat with the mandible, the nasal septum and the anterior portion of the cerebrum removed to show the origin, course and termination of the vomero-nasal nerves.

tion. He notes that the nerve to the organ in snakes leaves the anterior region of the rhinencephalon, passing directly downward to the upper wall of the organ surrounding it by deviating into thick medial and lateral branches. If one follows this nerve of Jacobson caudally by means of frontal sections it is seen that it is in close relationship to the medial aspect of the rhinencephalon, whereas the olfactory nerves proper enter the lateral and lower sides. Mihalkovics did not recognize an accessory olfactory lobe with which he was in all likelihood dealing and which will be discussed somewhat more fully later. He notes that the connection of Jacobson's organ with the nose is by way of a narrow cleft, hence odorous substances from the mouth could not get into the organ easily.

Seydel thought that in amphibia and turtles the organ existed to test expiratory air currents. Mihalkovics, because of the findings in snakes, cannot agree with this and is struck by the fact that where Jacobson's organ is highly developed it seems to be well protected. He feels that the organ exists to help the animal recognize sex among its own kind and notes that in birds, where vision replaces smell as a means for recognizing sex, Jacobson's organ is absent in the adult and the organ of smell in general is rudimentary.

Nemours (1930), in his comparative anatomic studies on the accessory sinuses in reptiles and birds, notes among other things the presence of Jacobson's organ in an adult oak snake (*Elaphi obsoleta confinis*). Here, as in other members of this family, the organ is located just lateral to the septum at its lowest point. It rests above the premaxilla, medially to the intermaxilla and above the vomer. The paraseptal cartilage projects in the lower inner wall of Jacobson's organ, giving the lumen a half-moon appearance. This knoblike projection of the cartilage, Mihalkovics suggests calling *eminencia fungiformis*. That part of the epithelium covering the fungiform eminence is respiratory in character. There are cilia present, according to Mihalkovics. On the side covered by sensory epithelium there is toward the lumen a layer of sustaining and olfactory cells. Then comes a deeper layer of cells, separated from the superficial layer by a capillary network, as demonstrated by Mihalkovics by special staining methods. The cells in the deeper layer are curious, arranged in strands, radially (*drüsenartige Zellstränge*, Leydig), which have in *Coluber natrix*, a species of adder studied by Mihalkovics, a length of 180 to 200  $\mu$ . and a width of 80 to 90  $\mu$ . There are no signs of excretory ducts, though Born thought that this tissue was glandular, but Mihalkovics, Leydig and Nemours feel that the cells are modified nerve cells.

In the suborder of Lacertilia (lizards), Nemours (1930) studied the adult chameleon (*anolis carolineus*). He states that "Jacobson's organ appears as a U-shaped structure beneath the ventral side of the anterior portion of each nasal cavity. A thick layer of olfactory type of epithelium is present on the medial surface and a thinner, nonciliated stratified type on the lateral surface."

The structure of Jacobson's organ, states Nemours, is different from that described for other species of the lizard. In the first place, the organ has two openings, one communicating with the nasal and one with the oral cavity. The nasolacrimal duct empties between the two openings. These two openings, it is clear, allow a more rapid diffusion of odorous bearing gases than just a single one would.

Crocodiles in general are not known to have, in adult stages at least, an organ of Jacobson. Sluiter (1892) reports the presence of a well developed organ in *Crocodilus porosus*; Röse (1893) does not agree but finds evidence in the same animal, in embryonal life, of such a structure. Howes (1891) also wrote on this topic.

Nemours (1930), studying a young alligator (*Alligator Mississippiensis*), found no trace of a Jacobson's organ in young or adult forms.

In the order of Chelonia, consisting of turtles and tortoises, there have been a number of students. Seydel (1896) made a lengthy contribution; Mihalkovics (1898-99) added his findings but disagrees with Seydel as to which structures are to be considered Jacobson's organ. McCotter (1917) agrees with Seydel, and Zuckerkandl (1910) and proves his point by establishing certain criteria. He studied *Chrysemys punctata*. Nemours (1930) studied and described the adult of another species, *Pseudemys elegans*. McCotter shows definitely that the olfactory area proper is separate from the neuro-epithelium of Jacobson's organ, that the olfactory fibers which supply the latter are separate from the nerves of the former and end in an accessory olfactory bulb.

Jacobson's organ is present in all orders of mammals and is best developed in the lower orders, such as Monotremes and Marsupials. In the primates so far examined it is said to be absent, at least in the adult. In man it is rudimentary and it has not been seen in Cetacea (whales). There are duly recognized variations in development in the different orders and even in closely related groups a remarkable constancy in the general structure of one organ and in its relations to the surrounding parts.

It appears typically as a bilateral tubular structure lined with mucosa, blind posteriorly, situated in the anterior ventral part of the

nasal septum, near the maxillary and premaxillary bones and opening in front in the region of the nasopalatine canal. The transverse section is seen to be either round or oval, and at times, because of surrounding structures which bulge into the organ, even crescentic or kidney shaped in outline. It is said to be better developed in small animals than in large ones, and where it is well developed it is closely surrounded by glands, first pronounced as serous by Klein (1881), whose ducts empty their secretion into the organ, as a rule posteriorly, but in some forms anteriorly too. Along the lateral wall is a plexus of blood vessels of cavernous structure, and supporting the whole is a more or less curved hyaline cartilaginous plate. Broom (1897-1898) has made a most careful comparative anatomic study of the cartilages surrounding Jacobson's organ in mammals. He arrives at the following important conclusions:

Though the organ varies in degree of development in even closely allied forms, throughout in large groups there is a marked constancy of type produced by the organ in its general relations and connections. For instance, the same cartilaginous arrangements are seen in all the Marsupials examined with but one exception; the two known Monotremes have a number of features in common different from all other animals. The same can be said of all rodents examined. Again in all higher Eutheria, where Jacobson's organ is well developed, there is a complex arrangement of the surrounding cartilages so unvarying that in forms such as *Pteropus*, where the organ is completely aborted, the cartilages continue to retain the same general arrangement. From this small tendency to variation in the cartilages and the organ a factor of value is present in the classification of Eutherian orders probably of more value than dentition or placentation.

A word must now be said about the nervous connections of the organ. Suspected almost from the beginning by most observers of being a sense organ, Balogh (1861) was the first to show that the epithelium contained typical olfactory cells. The staining methods in use at this time were unsatisfactory. These were the methods, however, used by Balogh (1861), Klein (1881) and Piana (1880). The advent of the Golgi technic permitted the question to be solved beyond all doubt, and the technic was adopted by v. Brunn (1892), v. Lenhossek (1892) and Retzius (1894) among others. At first thought by v. Brunn (1892) to be present only in the medial wall epithelium, it was demonstrated by v. Lenhossek (1892) in rabbits that olfactory cells did occur in the lateral wall. In addition to the olfactory cells characteristic supporting or sustentacular cells were

also demonstrated, proving conclusively that the olfactory portion of the mucosa was in no respect different from that of the olfactory area proper. The olfactory cells, furthermore, as in the olfactory area proper, terminated by way of an axone in fibrils of nerves undistinguishable from the regular unmyelinated olfactory nerves and were thought to be branches of these.

From Jacobson's time on it was known that the organ had, in addition to other nerve supplies, branches from what was thought to be the olfactory nerve. Leydig (1872) saw them; Balogh (1860-61) followed them to the brain. Read (1908) and McCotter (1912-17) did important work in this connection, confirming previous observations; Read traced the nerves carefully to the region of the median surface of the olfactory bulb, as did DeVries (1905). The former called these fibers coming from Jacobson's organ the vomeronasal nerves and so distinguished them from the ordinary olfactory nerves.

Balogh traced the nerve in the sheep, following it in the deep layers of the mucosa of the septum, through the cribriform plate to terminate in a small structure close to the olfactory bulb and designated by him as "Jacobson's hill." As McCotter (1912) calls attention to in his paper, he makes no mention of the histologic structure or the central connections of "Jacobson's hill."

G. Elliot Smith (1897), working with a fetal ornithorhynchus, described the olfactory bulb, and two ganglionic masses lying on it; one large and median receiving fibers from the olfactory nerves and one small and lateral receiving fibers originating in Jacobson's organ. He called this the "ganglion" of Jacobson's organ.

DeVries (1905) studying human embryos felt that the nerves coming from Jacobson's organ were identical with the nervus terminalis so prominent in lower forms.

Döllkin (1909) used Cajal preparation in very young embryos of man, rabbit and mouse. He traced fibers from the organ of Jacobson to a ganglionic mass which he called "ganglion terminale."

McCotter feels and in all likelihood is correct in saying that Elliot Smith had before him the embryonic accessory olfactory bulb; that DeVries' material was scanty and that Döllkin, working at a stage before the formation of the olfactory bulb, could not clearly prove that his ganglionic mass was in reality the forerunner of the accessory olfactory bulb.

v. Gudden (1870) in rabbits clearly recognized the existence of a structure close to and resembling the olfactory bulb proper;

Ganser (1882) definitely reports the presence of an accessory olfactory bulb in the mole; Köllicker (1896) is the first to furnish illustrations of this structure. Ziehen (1897) described it in marsupials (*Pseudochirus*) as like the structure described by v. Gudden in rabbits; Cajal (1911) described the accessory olfactory bulb in the mouse, rabbit and guinea pig.

McCotter (1912) definitely clears the air. The *nervus terminalis* is not functionally related to Jacobson's organ. Its distribution and central connections are made clear by Herrick (1909), Brookover (1910), Brookover and Jackson (1911) and McKibben (1911). McCotter makes it plain, first that the olfactory cells in the neuro-epithelium of Jacobson's organ terminate in fibrils belonging to olfactory fibers of a special group, which end in an accessory olfactory bulb. His paper is most important; he worked with serial sections and dissections of the structures in question in the opossum, sheep, bat, rat, guinea pig, cat and dog, as well as two reptiles (gila monster and snake). He summarizes much as follows:

"The accessory olfactory bulb is a ganglionic mass for the reception of fibers from the vomero-nasal organ and centrally gives off fibers that join the lateral olfactory tract. It is entirely separate from the olfactory bulb, though contiguous to it, being always on its dorsocaudal surface. Its size varies directly with the size of the vomero-nasal organ. It is evident that 'Balogh's hill,' Smith's 'ganglion' of Jacobson's organ, Döllkin's 'ganglion terminale' are the same thing as the accessory olfactory bulb."

The olfactory nerve filaments may be divided into two distinct groups, (a) ordinary olfactory fibers terminating in the olfactory bulb, and (b) vomero-nasal fibers terminating in the accessory olfactory bulb. The *nervus terminalis* probably belongs to a third group with entirely different central connections. McCotter suggests substituting the term *tuberculum vomero-nasale* for *bulbus olfactorius accessorius*, thus giving the vomero-nasal organ, the vomero-nasal nerves and the vomero-nasal tubercle as the component parts of this odd olfactory apparatus.

In 1917, McCotter undertook a similar investigation in amphibia (frog) and reptiles (turtle) and determined similar relationships in these forms. Herrick (1921) studied in the frog the connections of the vomero-nasal nerve and the accessory olfactory bulb, tracing fibers centrally from this last structure to the nucleus amygdala. This is the only detailed work done which attempts to determine the central relations of the vomero-nasal tubercle.

The cartilage always seen surrounding the vomero-nasal organ in mammals was first seen by Jacobson (1811-13), later by Stan-



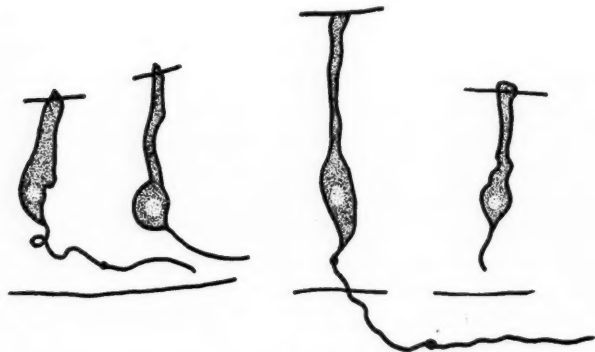


Fig. 7.

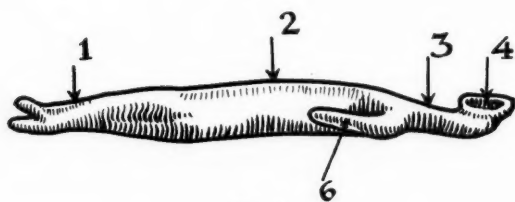


Fig. 8.

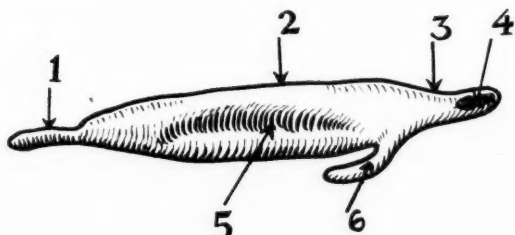


Fig. 9.

Fig. 7. Olfactory cells of the vomero-nasal organ in an embryo kitten (after Read). This type of cell resembling in every respect the sensory cells of the olfactory area proper is the important feature of the neuro-epithelium of Jacobson's organ. (Highly magnified.)

Fig. 8. (After Addison and Rademaker.) Drawing of a photograph of a model of vomero-nasal organ of a 21 day fetal albino rat; (dorsal view) anterior end of organ toward the right side of the page. The body (2) of the organ of Jacobson is seen to be roughly spindle shaped. The organ empties into the nose through the mouth (4) of a canal (3). Glands surrounding the organ empty into it by ducts anteriorly (6) and posteriorly (1).

Fig. 9. (After Addison and Rademaker.) Same features as in Fig. 8; this lateral view of outer side shows an indentation or groove on the lateral aspect of the organ (5).



nius (1854) and numerous others. It is hyaline in nature. Read (1908) states that in the cat the cartilaginous capsule is complete anteriorly and incomplete posteriorly; in the dog it is incomplete in its entire length. Brooms' (1895-97) investigations are the most complete.

Jacobson (1811-13), Klein (1881-82), Seydel (1895) and others noted the numerous glands surrounding and often emptying into the organ in mammals. Klein first pronounced the glands serous in nature.

The marked cavernous tissue development in many mammals about the lateral wall of the organ was noted by Piana (1880), Klein (1881-82) and Herzfeld (1889).

The manner of communication of the organ with the outside world varies. In most mammals the duct empties into the ductus naso-palatinus (sive incisivus Stenonis) ), thus establishing free communication with the oral cavity. In rodents, however, the duct as in man opens on the septal wall, directly into the nose.

The connection of the organ with the mouth was noticed in certain animals, i. e., horse (Jacobson, 1811); ass, camel, giraffe (Gratiolet, 1845); horse, camel, pig and ox (Minnett, 1925-26); horse (Kerkhoff, 1924), to be impossible because the nasopalatine canal is obliterated in its lower end.

In certain mammals, furthermore, cilia are demonstrable in the mucosa of the organ; on the side of the respiratory epithelium (Klein, 1881). Köllicker demonstrated, in calves at least, that the direction of ciliary action was from within outward.

Lastly, it may be said that in those vertebrates in which the organ cannot be demonstrated evidences, such as the cartilages accessory to the organ, remain to show its former presence.

The function of Jacobson's organ remains a puzzle to this day. Few workers have undertaken experimental procedures. Balogh (1860) filled the organ with tallow but arrived at no conclusion concerning its function; Mihalkovics destroyed it by cautery and noticed no difference in the feeding habits of the animal, although Broman (1920) asks pertinently why he did not observe reactions following the offering of noxious food.

Broman's (1920) experiments were the most complete. He worked with a wide range of animals. He felt, as a result of his observations (dog, guinea pig, etc.) that air is seldom present in the organ and that secretion (from the surrounding serous glands?) constantly escapes from them. He as well as others (Klein, 1881-

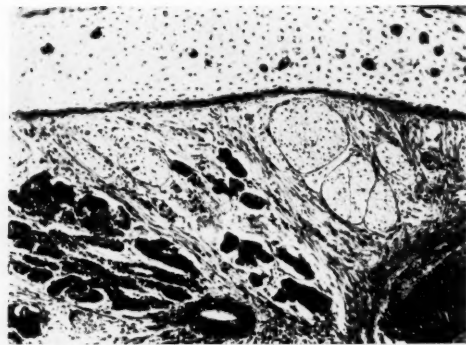


Fig. 10.



Fig. 11.

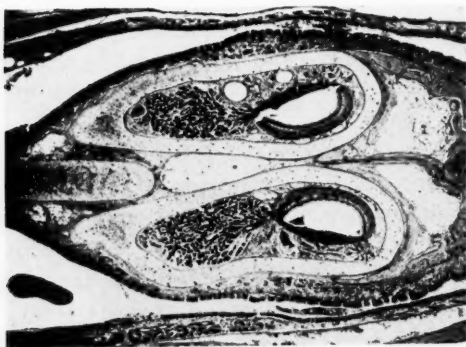


Fig. 12.

Fig. 10. Author's section (rabbit). (Photomicrograph.) Section through vomero-nasal organ illustrating vomero-nasal nerve bundles and glandular tissue. (High magnification.)

Figs. 11 and 12. Author's sections. Vomero-nasal organ in the rabbit showing, in Fig. 11, Jacobson's cartilage partly surrounding and in Fig. 12 completely surrounding the organ. In addition vomero-nasal nerve bundles, glandular and cavernous tissue are discernible. In the upper right hand illustration a gland duct is seen entering the lumen of the organ of Jacobson. (Photomicrographs. Low power.)

82), (Mihalkovics, 1899), noted the rich blood supply of the organ and the cavernous tissue about it, which led Broman to believe that Jacobson's organ was both erectile and contractile.

Because in so many animals the organ had a duct communication with the mouth, an obvious conclusion was that the organ existed to discriminate between useful and harmful substances (Cuvier). Jacobson himself thought the organ was a secreting apparatus but had doubts as to whether the secretion was a side function or whether the organ existed to discriminate in either taste or smell. The fact that in many animals the communication with the mouth was cut off even though the organ was well developed prevented a general theory that its function was olfactory in relation to food intake.

In many forms, too, the organ seemed well protected so that easy access of air is not possible. Nevertheless, the organ was obviously one with an olfactory function, for was it not supplied by branches from the olfactory nerve? v. Köllicker (1877) thought he had the answer, for if in many forms the oral cavity is cut off, and in others the protected position of the duct prevented easy access of air, then the organ must exist as a mechanism to test the animal's own juices brought by the rich vascular supply.

Seydel (1895) agreed with Broman that the organ seldom or never contains air, and the latter decided after his detailed study of morphologic and physiologic relationships that the fluid which always fills the organ by a pumping mechanism of one kind or other is alternately sucked in and expelled.

Herrick (1921) gives a critical resumé of Broman's labors, somewhat as follows:

"Broman states that though the pumping mechanism in mammals and reptiles is different in both it appears to be under control and to act rapidly. In lizards and serpents there is a mechanism by which the liquids of the mouth cavity can be forced in and out of the vomero-nasal cavity, thus providing an 'ideal mouth smelling organ.' In mammals the pumping apparatus is much more complex and diversified in different groups, in some species adapted to draw liquid olfactory media into the vomero-nasal organ from the mouth cavity, in others from the nasal cavity and in most cases from both these cavities."

Broman suggests that the liquid media derived from the respiratory passages of the nose enable Jacobson's organ to function in those mammals whose sense of smell is well developed (macrosmatic mammals) in "tracking" by odors (Spürsinn).

As Herrick says, however, it should be noted in this connection that the olfactory epithelium of mammals is not directly excited by gaseous media, as is sometimes taught, but the odorous substances

must first be dissolved in the liquid which always bathes the olfactory membrane. Nevertheless, it is not improbable that the liquid of the respiratory passages may absorb a larger amount of the odorous substances, and this more concentrated medium when sucked into the vomero-nasal organ would give to the latter an enhanced olfactory efficiency as a distance receptor.

Herrick observes further that Broman did not investigate amphibia. From Bruner's (1914) observations already cited it seems very probable that the aquatic "double smelling" amphibia first developed a lateral nasal diverticulum in close relationship to the choana especially adapted to serve as a mouth smelling organ, and that from this simple beginning the true vomero-nasal organs of anura and amniota have been derived. Herrick ends by stating that Broman himself reached a different conclusion, viz.: "that the organon vomero-nasale Jacobsoni is nothing other than the old water olfactory organ of vertebrates adapted for use on land," a position which Herrick justly finds untenable and which Broman unfortunately attempted to support by a reference to the N. terminalis, thereby revealing a total neglect of recent contributions to the innervation of this region. In this connection the works of Herrick (1909), Brookover (1910-1917) and McKibben (1911) are especially pertinent.

The latest contribution on the function of the organ of Jacobson is from the pen of Hamlin (1929). He had previously found that air could be expelled from the orifice of the organ in recently killed calves and rats by pressing the finger on the organ, toward the orifice while the heads were held under water. Seydel, of course (1896), had said that they never contained air and Broman (1920) that they seldom did.

Hamlin, further, while tracing out the blood supply of the organ, made what seemed to him a significant observation. He had filled the vessels of the organ, in rats, by injecting india ink into the aorta. Histologic examination, beside demonstrating the presence of the coloring matter, showed the lumen of the organ to be collapsed. It occurred to him that some factor of circulation, such as the pulse or blood pressure, might be the agency by which the contents of Jacobson's organ could be more or less continually ejected throughout the life of the animal. He thereupon examined living rabbits; under anesthesia and other precautions and controls, the orifice of one Jacobson's organ was exposed. Substances, such as adrenalin are then injected into an external jugular vein, while the orifice of the organ was observed by a microscope and the blood pressure

was recorded. He discovered that a bubble of gas or a drop of liquid (secretion, or some Ringer's solution added when the normal secretion seemed inadequate) appeared at the orifice of the organ when the effect of injected adrenalin was to elevate the mean arterial pressure. His data seems to leave no doubt as to the possibility of an increase in blood pressure tending to expel the contents of the organ. He would not say that this was the mechanism in the normal animal, nor as to whether higher pressures than normal are ordinarily required to expel the gas contents of the organ. Furthermore, he demonstrated that Jacobson's organ may "suck" liquid into themselves. After increasing the blood pressure by adrenalin injections and noting an ejection of fluid from the orifice of the organ it was seen that this fluid was sucked back in again when the blood pressure dropped. Hamlin feels that the cartilaginous capsule of the organ may have a twofold function, (a) it may act similar to the thoracic wall in respiration as a provision against collapse when its internal pressure is lowered following expulsion of its contents, either gaseous or liquid, and (b) it may serve to facilitate a rise in internal pressure by preventing an expansion of the organ at the time this pressure is being developed, thereby favoring the more complete ejection of its contents. In this way Jacobson's cartilage would facilitate both the introduction of fresh odoriferous material and its removal once the latter had served its purpose.

A few words as to Jacobson's organ in man may not be out of place at this time. As already stated, Ruysch (1703) was the first to observe and mention its occurrence in man. Sömmering figured it in man in 1809, and Meckel in 1820. Dursy described its development in the human embryo in 1869. In 1877, A. Köllicker made Jacobson's organ in man (embryo, child and adult) the subject of a special monograph and since then it has been studied in the human subject by others.

Köllicker gave the length of the organ in embryos as varying from 2 to 7 mm. Anton (1895) gives as its extremes 2.2 mm. and 8.4 mm. The canal averages about 1 mm. in width.

Mihalkovics (1899) investigated Jacobson's organ in a series of human embryos and found it always present. He states that when it cannot be found in a human embryo its absence is not the result of nondevelopment but due to early regressive changes. Grünwald (1925) and Peter (1925) state that the organ is practically always present in the newborn. Richter (1932) studied twelve embryos and fetuses and found it present in all but one, namely, a 9-month-old fetus. Whether it was really absent or had already regressed



Fig. 13.

Fig. 13. Author's section illustrating vomero-nasal organ in the rabbit and showing to the left cavernous spaces and to the right the medial wall of neuro-epithelium with nerve bundles just inside the cartilage. (Photomicrograph. Moderately high magnification.)

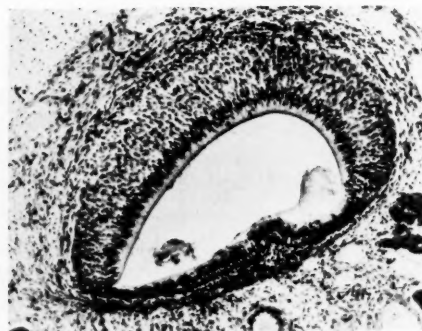


Fig. 14.

Fig. 14. Author's section illustrating vomero-nasal organ in the rabbit. The epithelium of the lateral (left wall) is respiratory in type and low. The neuro-epithelium forms the medial wall (right). A clear zone is seen next to the lumen, next a zone of dark staining oval nuclei of the sustentacular cells and then a broader band of lighter staining oval nuclei of the olfactory cells.

and disappeared he could not say. In twenty-seven children from the newborn to 5 years of age, it was macroscopically present nine times. It begins to disappear markedly, he thinks, after the fifth year. His summary is as follows: Jacobson's organ is regularly present toward the end of fetal life; it is not present constantly at birth; it is very often absent after two years of age; unless one uses a very large amount of material one cannot say just at what age it disappears; we do not know, in a specimen with the organ absent at 5 years of age, whether it disappeared during the first, second, third or fourth year.

Potiquet (1891) states that Jacobson's organ is not only present in the human embryo but that it can very often be found in the adult. He states that Jacobson's organ is easily found in the human cadaver as a wide crypt; the younger the subject and the fewer the mucosal lesions the greater the ease with which it is found. The orifice of the canal can be found in the living if sought for with care. Potiquet found it without trouble. It is situated just above the elongated ridge running from in front backward and constituted in great part by Jacobson's cartilage (Huschka's cartilage; paraseptal cartilage), which occupies the antero-inferior part of the septum.

Potiquet also states that according to the maximum and minimum measurements of Köllicker a lozenge may be outlined in the septum 7 mm. from the bottom to top, 8 mm. from side to side. This lozenge is the zone of search and within the orifice of the organ, if present, will certainly be found.

Seeing the facility with which the opening of the organ can be found in the cadaver it is astonishing how difficult it is to find it in the living subject. Previous to Potiquet, Moldenhauer appears to have been the only investigator who deliberately set out to find Jacobson's organ in the adult human subject but he failed to find it. Potiquet systematically sought the organ in 200 adults and found it about one time in every four attempts. In searching for Jacobson's organ in the human adult Potiquet states that artificial light is not good; also that pathologic conditions of the mucous membrane, as well as irregular undulations of the cartilaginous septum interfere with the search. The narrowness of the orifice of the organ protects it from accidental penetration by the probe, but when the probe does enter no pain is felt.

Anton (1895) searched for Jacobson's organ in seven human adults, three men (ages 35 to 61 years) and four women (ages 25 to 64 years). In three of these individuals the organ could not be found, possibly, Anton thought, because of catarrhal changes.



Mangakis (1901-2) reports finding Jacobson's organ in a young soldier. It was developed on each side of the septum and the orifices were very distinct. Each organ was 6.2 cm. in length. The nose in all other respects was normal and Mangakis was sure he was not dealing with a fistula.

Read says that in the human embryo (6 to 7 weeks old) the vomero-nasal organ of Jacobson is flattened from side to side (as in the case of the dog and the cat) and that the epithelium of the median wall is thickened. In a 4 months' human fetus the organ seemed to be circular in outline.

Parker (1922) states that in early infancy all humans show traces of a pair of organs that undoubtedly are homologues of the vomero-nasal organs of the lower vertebrate mammals.

According to Parker, each vomero-nasal organ in the newborn babe is a short tubular structure from  $\frac{1}{2}$  to  $2\frac{1}{2}$  mm. long and lodged in the lower anterior portion of the nasal septum. The organ opens into the nasal cavity by a minute pore on the free surface of the septum not far from its ventral border and only a short distance inward from the external naris. The tubular part of the organ extends posteriorly from this minute pore and ends blindly at a point somewhat higher than the level of the pore itself.

In early human embryos Parker says that the pore of the vomero-nasal organ can be identified easily on the medial face of the nasal chamber just within the anterior naris.

In human adults the organ, though commonly present, may disappear entirely. When present it occurs near the ventral margin of the nasal septum. As seen in transverse section the organ has the appearance of a tube flattened to the plane of the nasal septum. Its lateral wall is covered with an epithelium that resembles histologically the respiratory epithelium of the nasal cavity, and which may even be ciliated. The median wall is covered with an epithelium much like the olfactory epithelium except that well differentiated olfactory cells may not be present or may be completely absent.

The cavity of the organ is sometimes obliterated by excess epithelial growth and calcareous concretions may be present in the lumen. The organ in man is without a doubt rudimentary.

A few authors have called attention to the possibility of pathologic conditions arising in association with Jacobson's organ.

Potiquet seems to feel that the perforating ulcers of the nasal septum occurring as they do in the region in which Jacobson's organ





Fig. 15.



Fig. 16.

Figs. 15 and 16. Author's sections. Human embryo 23 mm. crown rump length. Sections through the nasal fossæ shown, in Fig. 15, the beginning indentation, and in Fig. 16 the complete formation of Jacobson's organ. The cross section of the organ in Fig. 16 is seen to be round. The palatal processes have not yet met, they point downward and the tongue projects upward into the oral cavity.

is situated are possibly due to bacteria entering and multiplying in the blind cul de sac.

Hajek (1890) thought that these perforating ulcers were due to bacteria attacking among other structures Jacobson's cartilage.

The fact that perforating ulcers and, as Potiquet lays stress on, tuberculous and syphilitic lesions, have a predilection for the region of Jacobson's organ and cartilage makes Potiquet feel that these conditions might possibly be recognized early as a "Jacobsonitis," a point of view possibly a little far fetched.

In 1925, Levin reported a cyst of the nose, which he considered possibly as springing from Jacobson's organ because of its medial position and close relation to the incisive foramen.

#### CONCLUSIONS.

The organ of Jacobson occurs probably in fishes, surely in amphibia, reptiles and mammals. It varies a good deal in its development, being absent in some adult forms, such as the birds, and atrophic in others, such as man. When absent or atrophied, evidences of its presence are usually seen in embryonic life.

The organ is part of an olfactory system complete in itself. This system consists of an end organ, the vomero-nasal organ, containing a neuro-epithelium, the chief component of which is a typical olfactory cell.

The olfactory cells end in axones going to form a special group of olfactory fibers, the vomero-nasal nerves.

The vomero-nasal nerves end in an accessory olfactory bulb best designated, according to McCotter, as the tuberculum vomero-nasale.

The precise function of this curious olfactory organ is not known.

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LXII.

TONSILLECTOMY IN THE TUBERCULOUS: INCIDENCE  
AND PATHOLOGY OF TUBERCULOSIS OF  
THE TONSILS IN ADULTS.\*

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There has long prevailed a very conservative attitude toward tonsillectomy in the tuberculous. This is based upon the fear of causing a lighting up of a quiescent pulmonary condition or of producing an acute local lesion at the site of operation. Other deterrent influences include the possibility of pulmonary complications due to aspiration of blood or oral secretions; a serious lowering of the patient's resistance through hemorrhage, or through interference with his nutrition from nausea or prolonged dysphagia. A further danger is thought to exist in the possibility of spreading a localized chest lesion by breaking down existing barriers, through violent physical effort incident to struggling or coughing during or following the operation.

At Glen Lake Sanatorium, the 660 bed tuberculosis hospital of Hennepin County, Minnesota, since 1919, tonsillectomy has been performed on 324 tuberculous patients, including 100 children. Of this entire group only one, operated on prior to 1926, showed an unfavorable course clinically. Since 1926, four cases gave X-ray evidence of increased pulmonary involvement without unfavorable clinical signs. We have observed that as a rule these patients have shown postoperatively the same degree of general improvement as have nontuberculous, tonsillectomized patients. Based on this experience, we venture to express the opinion that the tuberculous patient whose progress toward recovery is hindered by pathology of the tonsils should not arbitrarily be denied the benefits of a tonsillectomy because he has tuberculosis. With proper reservations he should be

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\*Presented before the annual meeting of the American Laryngological Association, Cleveland, Ohio, June 8, 1934.

given the benefit of the doubt. In these cases the tuberculous lesion in the tonsils is latent, the operation being indicated because the tonsils, as in the nontuberculous, constitute a source of focal infection which hinders his recovery. We do not discriminate against the tuberculous patient with a chronic focal infection in his teeth or sinuses. Why should we do so if there is evident pathology in his tonsils?

The highest percentage of favorable results in operating on pathologic tonsils in the tuberculous, we believe can be attained only by (1) a very careful selection of patients for operation; (2) by extreme care in employing an operative technic which will most effectively safeguard him from the possible complications suggested above; (3) by observing a carefully planned program for his after-care.

In our work the following principles have been consistently observed:

1. Tonsillectomy is advised only for those patients in whom the operation is definitely indicated, that is, the presence of such pathology and symptoms as would demand enucleation in a nontuberculous individual. No patient in a hopelessly advanced stage and no patient, except in extreme emergency, is operated on while his tuberculosis is in the acute state. Emergency cases would include those with dysphagia, severe irritative cough or vomiting due to pathologic tonsils which thus interfere with recovery.

2. Preoperative treatment includes rest in bed for an indefinite period of time, during which interval the patient is carefully observed as to temperature, cough, sputum, weight, chest condition and bleeding and clotting time. In anticipation of operation the patient is taught how to relax so as to minimize gagging, straining and coughing. Fear of the operation is dispelled as far as possible, and the patient is impressed with the importance of co-operation with the operator. Calcium lactate, more recently calcium gluconate, is administered preoperatively whenever indicated.

3. Preoperative sedatives (as luminal, allonal, etc.) are given only when necessary to relieve undue nervousness. Superficial anesthesia is secured in patients with a hypersensitive pharynx or hypopharynx, by swabbing with a minimum amount of 8 per cent cocaine. One per cent novocain with suprarenin, freshly prepared in the presence of the operator, is slowly injected.

4. The operator is seated lower than the patient, causing the latter to incline his head forward and downward, so that during the

operation all drainage is forward, thus avoiding the accumulation of saliva and blood in the back of the mouth cavity where it can cause irritation of the epiglottis or larynx, or be aspirated. This we regard as a very important point, often overlooked.

5. Should any bleeding occur it is immediately controlled by pressure applied by means of a hard sponge, by a hemostat, or by ligation with triple O catgut on a fine half-curved cutting needle.

6. Blunt dissection and the slowly closed Bruenings snare, as modified by one of us, are routinely used to lessen the chances of bleeding. With children gas induced ether or, more recently, chloroform anesthesia, a modified Sluder method and the Rose position are employed.

7. The after-care is all important. This includes rest in bed for at least four weeks, special attention to minimize postoperative sore throat, frequent nourishment, beginning immediately after the operation, to keep up both resistance and body weight, and the careful guarding against postoperative bleeding and vomiting, with their untoward effects.

It is our belief that under these conditions the tuberculous patient is at least as good a surgical risk as the average ambulatory case.

Collapse therapy, now used so extensively in the treatment of pulmonary tuberculosis, is not a contraindication in tonsillectomy.

Tuberculosis involving the palatine tonsils has been recognized as a pathologic and clinical entity even prior to the isolation of the tubercle bacillus. Cornil,<sup>1</sup> in 1875, and Deplous,<sup>2</sup> in 1878, were among the earliest authors to report the observation of tuberculous ulcerations of the tonsils and pharynx. The diagnosis in these cases was made by clinical examination. Orth,<sup>3</sup> in 1879, was able to produce in rabbits tuberculosis of the tonsils and adjacent cervical lymph nodes by feeding them tuberculous tissue. His work was adequately controlled, and was, we believe, the first absolute proof of one definite means by which the tonsils could be infected. Reports by Strassman,<sup>4</sup> Dmochowski,<sup>5</sup> Kruckmann,<sup>6</sup> Walsham,<sup>7</sup> Schlenker,<sup>8</sup> Ruge<sup>9</sup> and others soon followed. These authors discussed especially the frequency with which tuberculosis was found in tonsils removed at autopsy from patients who had died of advanced pulmonary tuberculosis.

The incidence of tuberculosis of the tonsils in routine tonsillectomies, as reported in the literature, is listed in table I. The diagnosis in these cases was usually made by examinations of microscopic sections, and most authors included both adults and children in their

statistics. For the entire group of 30,676 an average incidence of 2.03 per cent was found. The higher percentages were, as a rule, found in the older literature and this corresponds with the higher general incidence of tuberculosis at that time. The general opinion at the present time is that 1 per cent of routine tonsillectomies will reveal tuberculosis when the tonsils are examined microscopically.

The incidence of tuberculous tonsils in adults who have active pulmonary tuberculosis has not been as well established, except in those patients in whom death ensued and the tonsils were removed for examination at autopsy. Mullin,<sup>19</sup> in a series of twenty-five living patients known to have pulmonary tuberculosis, found ten (40 per cent) whose tonsils revealed tuberculosis. Heaton<sup>24</sup> reports finding

TABLE I.

INCIDENCE OF TUBERCULOUS TONSILS IN ROUTINE TONSILLECTOMIES.

AUTHOR	No. CASES	TBC	
		TONSILS	PER CENT
Weller (10)	8,697	204	2.35
Wilkinson (11)	10,000	52	0.52
Crowe, Watkins and Rothholz (12)	1,000	46	4.60
Judd (13)	1,000	23	2.3
Sewall (14)	772	30	3.9
Barnes (15)	150	3	2.0
Lewin (16)	200	9	4.5
Robertson (17)	232	19	8.0
Willis (18)	108	9	8.0
Mullin (19)	400	17	4.25
Hodenpyl (20)	200	1	0.5
Morris (21)	2,321	39	1.7
MacReady and Crowe (22)	3,260	138	4.23
Cornell (23)	2,336	35	1.49
Total	30,676	625	2.03

thirteen (22 per cent) tuberculous tonsils in a group of fifty-nine adults with active pulmonary tuberculosis. Camp<sup>25</sup> reports a series of twenty-seven patients, in twenty-five of whom a definite diagnosis of pulmonary tuberculosis was made. Nineteen (70 per cent) of this group had tuberculous tonsils. Wood<sup>26</sup> found 5.2 per cent of 1871 tonsillectomies were tuberculous. However, in those patients with pulmonary tuberculosis the tonsils were tuberculous in 69 per cent.

The tonsils removed from 112 adult patients in residence at Glen Lake Sanatorium were subjected to microscopic examination. The results are listed in table II. Cases in which only tonsillar tags or a single tonsil were removed have not been included. The relationship

between the stage of pulmonary pathology and the percentage of tuberculous tonsils is also charted in the same table. One sees immediately that the incidence of tuberculous tonsils is much greater among those patients with the more extensive pulmonary lesions. Fifty-four of the patients had far advanced pulmonary tuberculosis and in this group thirty-one (57.4 per cent) had tuberculous tonsils. Thirty-four had moderately advanced tuberculosis and fifteen (44.1 per cent) of these had tuberculous tonsils. The lowest incidence percentage occurred in the group with minimal lung pathology, where only two (16.6 per cent) of twelve had tonsillar tuberculosis. In

TABLE II.

INCIDENCE OF TUBERCULOUS TONSILS IN RELATION TO STAGE  
OF PULMONARY TUBERCULOSIS.

STAGE DISEASE	NO. CASES	NO. TONSILS	
		TBC.	PER CENT
Far advanced .....	54	31	57.4
Moderately advanced .....	34	15	44.1
Minimal .....	12	2	16.6
Nonclinical .....	4	0	—
Normal chest .....	8	0	—
Total .....	112	48	42.8

the group with nonclinical lesions or no pulmonary tuberculosis, twelve in all, the tonsils were negative for tuberculosis in all twelve cases. For the entire group the incidence was 42.8 per cent. If the cases with nonclinical and no pulmonary tuberculosis are eliminated, the incidence in the 100 cases with active pulmonary tuberculosis would be 48 per cent.

Through the courtesy of the University of Minnesota Hospital we were able to obtain for microscopic study the tonsils removed in a series of 100 adult routine tonsillectomies. The purpose of the control group was to eliminate the possibility of mis-diagnosis, as suggested by Weller,<sup>27</sup> by interpreting foreign body pseudotubercles as true tubercles. This group was used as a control series for incidence in the general adult population. Only one case with tuberculous tonsils was found, and we were surprised to find this one positive case was an ex-patient from Glen Lake Sanatorium.

Tuberculosis of the tonsils is usually described as a "latent" lesion because it is rarely diagnosed by gross examination. In this series there were no gross ulcerative lesions. Isolated cases of this type of lesion have been reported by Latham,<sup>28</sup> Oertel and Griot,<sup>29</sup> Brown

and Doig,<sup>30</sup> Doyle,<sup>31</sup> Horgan,<sup>32</sup> Murray and Maxwell,<sup>33</sup> Jacques,<sup>34</sup> Dickey<sup>35</sup> and others.

The microscopic lesions usually found can be divided into two major groups. The first group is composed of those in which the tubercle formation is localized and in close relation to the cryptal epithelium. This constitutes the majority of cases. The lesions may be bilateral, but very frequently they are unilateral. The second large group is composed of those in which the tubercles are disseminated throughout the stroma of the tonsils. The germinal centers usually escape involvement. These disseminated lesions are most often bilateral.

The tubercles themselves are epithelioid in type and many of them are composed entirely of epithelial cells. Giant cell formation is fairly common and was present in forty-six of the forty-eight positive cases. Caseation, on the other hand, is not a marked feature, which probably accounts for the rarity of the ulcerative type of lesion. Caseation was present in twenty-two cases. Tubercles were found in both tonsils in twenty-eight instances, an incidence of 58.3 per cent.

No attempts were made to isolate tubercle bacilli by direct smear, cultures or animal inoculation.

We have long felt that the actual incidence of tuberculous tonsils was probably higher in our series as well as in those reported by other authors, in which microscopic examination was made of one or, at the most, two sections from each tonsil. Sewall<sup>14</sup> reported the results of serial sections of forty-three tonsils. One tonsil was found to be tuberculous, which had been negative on previous microscopic study. These sections, however, were on routine cases in which the incidence of tuberculosis in the entire series was low. In our series the tonsil tissue was all saved so it was possible for Dr. Peterson, working in our laboratory under the direction of one of us, to make serial sections of twenty pairs of tonsils which had previously been reported as negative for tuberculosis. In four of these twenty cases he found tubercles by this method. This indicates that the results in the usual microscopic examination are too low and constitutes a definite percentage error when computations are made from them. These positive cases found only by serial section were not included in the figures tabulated in table II. The reported figures represent only those cases which were found to be positive by the usual microscopic studies.

The route by which the tubercle bacilli reach the tonsil has been a topic of controversy for years. Theoretically and anatomically

there are five possible routes to consider: (1) inhalation, (2) ingestion, (3) retrograde lymphatic drainage from tuberculous cervical adenitis, (4) hematogenous, (5) tubercle bacilli in the sputum. The first two routes would represent a primary involvement of the tonsils, while the latter three would indicate a secondary infection. It is our opinion that the infection of the tonsils, at least in the series reported, was secondary to the pulmonary involvement. Grober<sup>36</sup> presented anatomic studies to prove that tubercle bacilli could invade the tonsil and then descend to the lung pleura via the cervical lymphatics. Van Zwaluwenberg and Grabfield<sup>37</sup> published a clinical study tending to confirm the work of Grober. In their series of sixteen tuberculous tonsils they found that fifteen showed a pleural cap when examined by X-ray. In twenty-seven cases where the tonsils were nontuberculous, only three showed this pleural cap. As a result of this study the authors concluded that the tuberculosis was primary in the tonsil and secondary in the lung. The authors also felt this route of infection explained why the lung apices were usually first involved in pulmonary tuberculosis. Our findings tend to prove the tonsillar involvement to be directly proportional to the degree of lung pathology. This could hardly be true if the tonsil infection were primary and the pulmonary lesion secondary.

It is our opinion that in the majority of cases the tonsil becomes infected by means of the sputum loaded with tubercle bacilli. We do believe the tonsils may be infected by the hematogenous route, but this is the exception rather than the rule. The pathologic picture cannot give the answer to this problem, as Krauspe<sup>38</sup> and Otto<sup>39</sup> have both shown that, except in very early cases, the differentiation between the resorptive and hematogenous involvement is extremely difficult if not impossible.

In our series there was a definite relationship between positive sputum and tuberculous tonsils. In the 100 cases of active pulmonary tuberculosis reported, seventy-one had a positive sputum found by direct smear or guinea pig inoculation. Of these, forty-one (57 per cent) had tuberculous tonsils. In the remaining twenty-nine cases no positive sputum could be found. Of this group, seven (24 per cent) had tuberculous tonsils. In other words, tuberculous infection of the tonsils occurred between two and three times as often in the positive sputum group as in the negative sputum group, although both groups had active pulmonary tuberculosis. Therefore we hold to the opinion that while hematogenous secondary tonsillar involvement can and does occur, it is not usual. We believe infected sputum is the causative factor, at least in the series here reported.



## CONCLUSIONS.

1. The incidence of tuberculosis of the tonsils in 112 adults was 42 per cent. If the twelve cases without pulmonary tuberculosis were eliminated, the incidence would increase to 48 per cent.

2. Serial sections of the excised tonsils would materially increase the incidence. This was demonstrated by such a study of twenty pairs of tonsils, in four of which tuberculosis was found. These had previously been reported negative by the usual microscopic study.

3. In a control series of 100 routine tonsillectomies, the incidence was 1 per cent, the positive case being found in an individual who had previously been treated for active pulmonary tuberculosis.

4. Tuberculosis of the tonsil is a "latent" lesion, in that rarely is there enough destruction of tonsillar substance to cause "gross" or ulcerative lesions.

5. The tubercles in the tonsil are usually in close proximity to the cryptal epithelium.

6. The tubercles are primarily epithelioid in type, with giant cells nearly always present. Caseation is found in about half the cases and is not marked.

7. The tonsils, in our opinion, become infected by tubercle-laden sputum secondary to the open tuberculous pulmonary lesion.

8. No case of primary tonsillar tuberculosis could be demonstrated in this series.

9. Tonsillectomy in the tuberculous when the patients are carefully selected and prepared, are safeguarded by a proper operative technic and are given the postoperative care demanded by their condition, presents no special hazard which would justify failure to perform tonsillectomy when indicated.

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### LXIII.

## INFECTIONS OF THE TEMPORAL BONE WITH SECONDARY MANIFESTATIONS.\*

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This presentation is based on a personal review of three hundred and eighty-four case histories. All patients were under ten years of age. Three hundred and forty-five were five years or under. A total of five hundred and sixty-one mastoid operations were performed. Bilateral mastoid operations were done on one hundred and nine of the patients and secondary mastoids on sixty-two. The temporal bones were secured from nine of the patients who died.

The parallel clinical and pathologic sequence in otitis with associated diseases will be followed. Gastro-intestinal disturbances associated with otitis will receive major consideration.

Upper respiratory infection is the usual cause of otitis in the infant. Debilitating diseases, congenital defects, and deficient constitutional resistance contribute as predisposing causes of recurrent mastoiditis.

The membrana tympani deserves primary consideration. Its strength is important as a contributing factor causing mastoiditis. Its appearance is most important in the diagnosis of mastoiditis. The membrane is sufficient to withstand the expanding pressure of pus in the middle ear, thus causing the fluid to extend through the aditus to the mastoid antrum. The fact that forty-five of our mastoids operated upon had intact membranes and that twenty-five of this number were subperiosteal abscesses serves as evidence of the resistance inherent in the structure of the tympanic membrane. Recurrent otitis causes thickening of the membrane, thereby increasing its resistance to pressure. The efficacy of this resistance is again demonstrated by the number of cases of secondary mastoiditis which present postauricular swelling with intact tympanic membranes.

Changes in the appearance of the membrana tympani furnish valuable indications of the invisible processes in the mastoid. When the picture becomes vague, it is most difficult by other means to

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\*Read at the annual meeting of the American Otological Society, Inc., April 6, 1934, Atlantic City, N. J.

estimate obscure destruction. While it is true that minute changes in the membrane have been present in all the cases of infant mastoiditis I have examined, it has been impossible in some cases to differentiate between the membrane housing a slowly resolving mastoiditis and one associated with progressive destruction. These patients present real problems and demand most careful investigation by the otolaryngologist and pediatrician. The resilient membrana tympani will return to a nearly normal appearance, but the mastoid antrum may still contain a low-grade infection. The aditus, being occluded, prevents drainage and aeration of the antrum cavity.

When the active signs of mastoiditis are present, the diagnosis is simple, but after the discharge from the middle ear has ceased mastoid tenderness has disappeared and the fundus approaches normal the diagnosis is difficult. If the patients having obscure mastoiditis are subjected to operation the following pathologic conditions may be present: (1) The antrum is filled with a pale, gelatinous mass. If the adjoining cells have started to form, they contain the same material; (2) By removing the cortex with care, it is possible to uncover soft tissue resembling a thickened and diseased sigmoid sinus wall. Further investigation proves the tissue to be a thick, encapsulating membrane containing necrotic material and occasionally thick, creamy pus. In these cases the antrum is large and the aditus obstructed; (3) When the antrum cavity is entered, one is immediately impressed by the extent of cavitation. The lining membrane presents a dirty-white appearance resembling the pyogenic membrane found with cholesteatoma. Caseous or flaky white material may be present; (4) The last picture to be described is one which on casual observation one might interpret as normal. In these cases the cavitation has progressed to such an extent that the enlarged antrum occupies nearly all of the mastoid process. The bone surrounding this cavity has no membranous covering and is porous in structure. Except for excessive paleness, it might be considered cancellous bone. It is soft in character and can easily be wiped away with gauze on a curette. There is little or no vascularity, judging from the "dead" appearance.

The invasion of a superimposed acute infection would be confronted with feeble resistance.

Gastro-intestinal cases with obscure ear pathology usually have one of the above conditions revealed at operation. If an added acute infection is present, pus will also be evident.

The various conditions described may also be found by necropsy. It is understandable, therefore, why some observers have classed

these conditions, especially No. 4, as "terminal pathology." Histologic preparations prove their chronicity.

Myringotomies rarely have more than a transient effect on protracted cases. Frequently vomiting will cease, feedings improve and occasionally the weight will increase, but in our experience the eventual recovery was delayed. A few cases ended fatally because of surgical procrastination. Myringotomies may result in profuse bleeding, due to excessive granulation tissue in the middle ear. An absence of discharge may mean an occluded aditus.

The necessity for repeated myringotomies is evidence of chronicity. The procedure is rarely effective.

One hundred and twenty-nine cases developed mastoiditis following myringotomy and one hundred and thirteen followed spontaneous rupture. The remainder had no definite designation. Only ten of the myringotomized cases had been incised before forty-eight hours from the onset of ear symptoms.

While no record of vomiting and gastro-intestinal irregularity was tabulated in the more acute cases, the frequency of their occurrence and the relief obtained by operation was noteworthy.

Vomiting in the fulminating stage and then later in the obscure stage, combined with our frequent observation of exudate in the labyrinth, seemed significant. The vomiting in meningeal involvement and gastro-intestinal cases with fulminating otitis or obscure destructive otitis may result from labyrinthine irritation.

After mastoiditis has apparently resolved but the membrana tympani has failed to resume its normal appearance, various gastro-intestinal symptoms may appear. Food refusal is manifested early; pallor is marked; the bowel movements become irregular and a failure to gain in weight is the result. As the disturbance progresses, nausea and vomiting occur and a diarrhea with dehydration ensues. Acidosis accompanies these symptoms. Physical examinations and laboratory data indicate no other organic changes. Changes in feeding formulæ offer no permanent relief.

Patients with mastoid involvement exhibiting the gastro-intestinal syndrome respond favorably following early surgical procedures. If the operation is delayed until the situation is considered hopeless, there is a much higher mortality. The course of recovery is rather constant. Vomiting ceases; the demand for food increases; bowel movements become regular, and as a result the weight improves. Even the fatal cases show some improvement following operation. Some of my failures have been due to premature closing of the mas-

toid incision. Patients who were doing nicely after their operation had a recurrence of symptoms with fatal results. More recently I have allowed the wounds to remain open with more favorable terminations.

Our necropsy records from 1926 to 1933, inclusive, produced twenty-seven cases of meningitis with ear involvement. If one hundred per cent autopsies were obtainable on infants under five years of age—regardless of the supposed cause of death—a histologic examination of the temporal bones would reveal a startlingly high percentage of protracted ear involvement.

Among the concurrent diseases, the most outstanding are the pneumonias with bronchopneumonia predominating. These cases numbered twenty. They seemed to fall in two groups: those occurring in the acute stage, either preceding or following the otitis, and those classed as terminal pneumonias occurring before death. These patients were subjected to mastoid operations when indicated, independent of any relation the otitis might have had. Local anesthesia was employed. The best results were obtained in those patients who failed to recuperate promptly from their pneumonic process. Five cases of facial palsy existed before the operation and were transient. Pyelitis numbered five. Medication was administered to these patients, so no deductions are drawn. They all recovered. Erysipelas occurred in four cases, the remainder having concurrent diseases as follows:

- Brain abscess—One.
- Pyelonephritis—One.
- Endocarditis—One.
- Acute hemorrhagic nephritis—One.
- Polio-encephalitis—One.
- Endocrine dyscrasias—Three.
- Multiple abscesses—Four (negative culture in blood stream).
- Parotid abscess—Two.
- Retropharyngeal abscess—Three.
- Rickets—One.
- General debility with a multiplicity of diseases—Two.
- Congenital heart—One.
- Mental deficient—One.
- Persistent hemostaxis—One.
- Pyelonephrosis—Two.
- Bacillary dysentery—One.
- One case of nephritis of long duration which resolved following the mastoid operation.

It will be noted in our statistical table that the deaths numbered twenty-six. Autopsies showing meningitis and ear involvement were twenty-seven. The discrepancy is due to undiagnosed antemortem ear pathology; therefore, they were not on the otologic service.

STATISTICAL TABLE.\*

Total number of patients	384
Total number of operations	564
Patients 5 years of age and under	340
Double mastoid operations	109
Single mastoid operations: left ear	140
Single mastoid operations; right ear	144
Sinus thrombosis (two cured by transfusion)	9
Secondary mastoiditis	62
Deaths	26
Spontaneous rupture of membrana tympani	113
Myringotomy (only ten within the first forty-eight hours)	129
Operative mastoids with unruptured drums	45
Tonsils present	274
Tonsils removed	73
Gastro-intestinal cases (eleven deaths)	45
Cervical adenitis	184
Cervical adenitis more pronounced on involved side	28
Cervical adenitis more pronounced on opposite side	3
Subperiosteal abscesses	68
Subperiosteal abscesses with unruptured drum	25
Streptococcus hemolytic	221
Pneumococcus (6 type 3, 1 type 4, 1 type 6, 1 type 5, rest untyped)	20
Autopsies (meningitis with ear involvement)	27

## REPORT OF CASES.

CASE 1.—M. H., 9 years, 6 months, was admitted February 27, 1930. The mother said the child was perfectly well until one week previously. The child complained of right sided frontal headache, worse at night; practically absent during the day. He vomited practically everything for six days. The temperature ranged from 100 to 103 degrees.

*Impression.*—(1) Brain tumor, (2) tuberculous meningitis, (3) encephalitis, (4) acidosis, (5) hysteria, (6) dehydration.

Lumbar tap on the 27th returned clear fluid; not under increased pressure. On the 28th there was no evidence of organic neurologic involvement. The vomiting was probably functional in type. The spinal fluid, examined on the 3rd, showed marked increase in cells. On the 3rd, there was a bilateral Kernig's sign and a diagnosis of meningitis was made. On the 5th, examination showed bilateral maxillary antrum involvement; on the 7th, paralysis of the left external rectus and paresis right external rectus. On the 8th, there occurred rigidity of neck, bilateral Brudzinski, right Kernig signs; upper abdominal reflex absent, lower sluggish.

Autopsy findings (March 21, 1930), were: Acute tuberculous basal meningitis; bilateral chronic fibrous pleurisy; chronic caseous and calcified lymphadenitis;

\*From the Otolaryngologic and Pediatric Service at the New York Post-Graduate Hospital:





Fig. 1. Exudate in the internal auditory meatus. Serous exudate in the scala tympani, basilar turn of the cochlea. Perivascular hemorrhage. Dilated and congested blood vessel. (Case 1.)

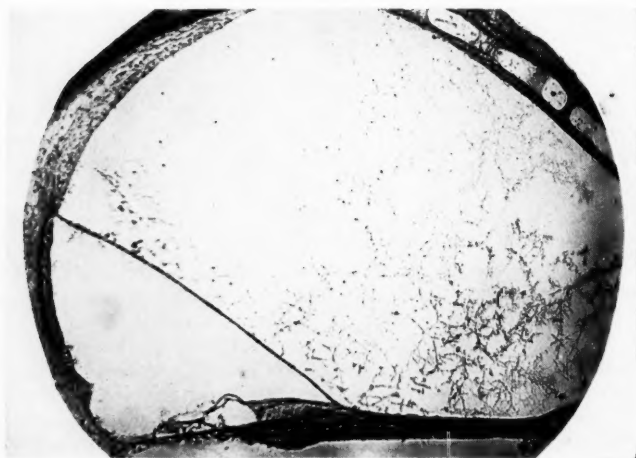


Fig. 2. Serous exudate in the scala media and serofibrinous exudate in the scala vestibulæ. (Case 2.)

chronic hemorrhagic colitis; acute purulent bronchitis; chronic bronchiectasis; chronic splenitis.

CASE 2.—D. S., 3 years, 7 months, was admitted January 24, 1932.

For the previous 20 days the child had had a cold. On January 14th it seemed weak and had a temperature of 104. Bilateral cervical adenitis appeared that day. From that time to the date of admission the temperature ranged from 99 to 106. There were redness of the throat; purulent material in the pharynx; pains in the ears and coughing.

January 24, 1932. The child had a chill, seemed delirious, and complained of pain in the knee, which was swollen. January 25th, diminished breathing and voice sounds were heard. There was restriction of motion of the right leg with a pseudo-Kernig sign and rigidity of the neck. The throat was reddened. The ears were pronounced negative. Blood culture on the 26th showed streptococcus. On the 28th a transfusion was given. Spinal tap on the 28th showed 104 cells and a positive culture of streptococcus. Antistreptococcic serum was administered intraspinally.

The clinical diagnosis was acute upper respiratory infection; bilateral cervical adenitis; septicemia (streptococcus hemolytic); bilateral otitis media and mastoiditis; meningitis; purulent bilateral lateral sinus phlebitis; purulent diffuse meningitis; cervical adenitis.

Autopsy showed: Bilateral otitis media; mastoiditis; purulent bilateral lateral sinus phlebitis; purulent diffuse meningitis; bilateral cervical adenitis; congestion of liver, spleen and kidneys; bronchitis; bronchopneumonia; streptococcus hemolyticus septicemia.

CASE 3.—E. F., 4 months of age, admitted January 11, 1932. The birth weight was seven pounds, ten ounces. Weight on admission was eight pounds and four ounces. The child had been breast fed for only two and one-half weeks, after which it was placed on artificial feeding. On the occasion of adding dextromaltose to the formula, at two and one-half months of age the child developed diarrhea lasting three weeks (having five to seven watery green stools a day). On November 28th, the child had abdominal cramps, turned blue, and held his breath.

This was a small, emaciated infant. The chest showed beading of the ribs. X-ray of the chest, taken before the child entered the hospital, showed a spherical mitral type heart, probably congenital. The weight during the first two weeks in this hospital remained stationary; the temperature remained normal. The stools appeared normal, and there was no vomiting. Eighteen days after admission the child developed a fever which on one occasion went to 105. Examination showed the throat red and both ear drums lusterless. Both drums were incised and a small amount of serum exuded. The following day a transfusion of 100 cc. of blood was given. The fever continued for the remaining nine days. The child vomited occasionally and had a mild diarrhea, passing four to five loose green stools every day. The formula was changed during the last nine days, but the child lost gradually one and one-half pounds. Five days before death the lungs showed a few fine crackling rales posteriorly and a few in the right apex anteriorly. The following day a second transfusion of 80 cc. of blood was given. X-ray examination of the lungs subsequently revealed a possible low-grade pneumonia on the right side. This condition became progressively worse and he died February 5, 1932, twenty-five days after admission.

The clinical diagnosis was malnutrition; bronchopneumonia.

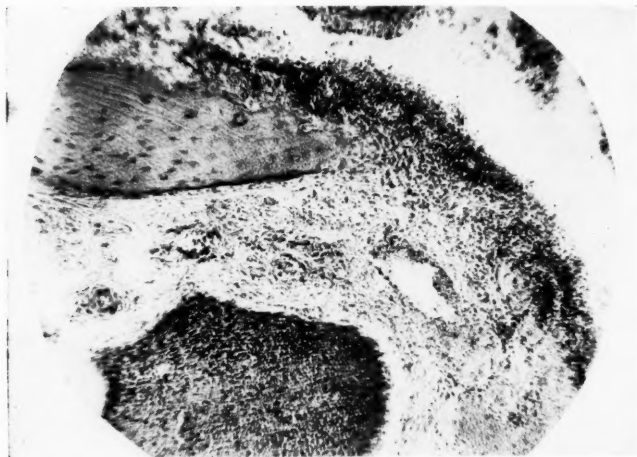


Fig. 3. Erosion of bone forming the fallopian canal with a dehiscence in the canal housing the facial nerve. (Case 3.)

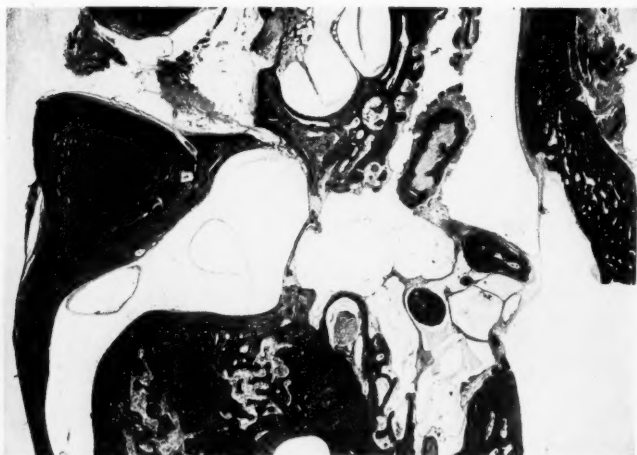


Fig. 4. Adhesions in the middle ear. Membrana tympani resolved. Fibrous thickening and exudate. Round cell infiltration. Mastoid mucosa thickened, infiltrated with polynuclear and round cells. (Case 4.)

On January 12, 1932, the drums were seen with difficulty; both were negative as far as could be determined.

On January 26, 1932, the drums were dull, lusterless and prolapsed. Operation was advised if there was any decrease in weight. On January 28, 1932, the right drum membrane was lusterless. The left ear was similar to the right, but there was some redness in the upper quadrant, without bulging. January 29, 1932, both drums were incised and serum obtained. January 30, 1932, eye examination showed, O. S., slight atrophy; O. D., negative.

On February 1, 1932, the ears were negative; irrigations were discontinued.

Autopsy showed: Left otitis media; left mastoiditis; bronchopneumonia; acute congestion of brain, liver, spleen, pancreas, lungs, kidneys and intestinal tract.

CASE 4.—M. C., 3 months old, was admitted on February 25, 1932. The weight on admission was six and one-half pounds. The child was breast fed during the first two weeks of life; then because of insufficiency of mother's milk it was given artificial feeding. Vomiting started at that time. It was always of a projectile type and consisted of practically the entire feeding. The child retained five out of eight feedings daily, with occasional days in which there was no vomiting. On February 27th, a transfusion of 75 cc. of whole blood was given. The temperature varied from 97 to 99.2 degrees, being elevated above normal on only two occasions. On February 29th, the right ear was normal, the left drum bulging. Myringotomy was done and pus obtained. The left ear was irrigated twice daily. There was marked malnutrition and dehydration; the skin was loose and very dry. The abdomen was moderately distended and doughy.

On March 2nd, at 8:30 a. m., six days after admission, the child was found very blue and died within a few seconds. Until this time the progress had been satisfactory.

The clinical diagnosis was: Marasmus; pylorospasm.

The postmortem findings were: (1) Diffuse foreign body reaction in both lower lobes; (2) hypertrophy of the pyloric ring; (3) congestion of the kidney, liver and spleen; (4) involvement of the thymus.

CASE 5.—L. P., seven months of age, admitted February 11, 1932. About twenty-four hours before admission the child began to cry and seemed irritable. On examination, the abdomen was tense and tender. There was a persistent fever. On February 12th, the face was flushed and showed evidence of pain. Moderately congested drum. There was exaggerated breathing of the right upper lobe. The abdomen was somewhat distended and very sensitive on palpation. The feet were flexed to the body.

The diagnosis was: Peritonitis; acute appendicitis; beginning right central pneumonia.

On February 12th, ears were negative. On February 14th, operation for acute diverticulitis was performed; the abscess was found. The temperature continued following operation and the child improved somewhat. On February 19th, the right ear was negative; the left ear slightly congested. On incision pus was obtained. Bilateral myringotomy was again done on February 25th and pus was obtained from both ears. On February 26th, the right ear showed an acute catarrhal otitis media, mild; the left ear showed the same, a little more severe. On February 27th the ears had ceased draining, and the possibility of performing a mastoidectomy was being considered. On February 29th, the ears were almost completely well. The

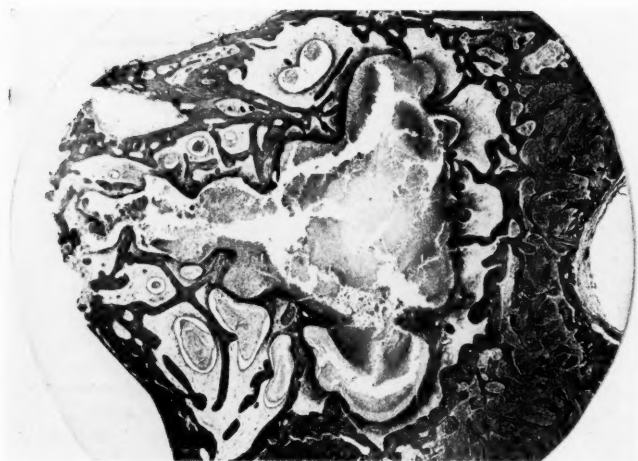


Fig. 5. Bone destruction. Involvement of the mastoid with cavitation. Thickened lining membrane, heavily infiltrated with polynuclear cells. (Case 5.)

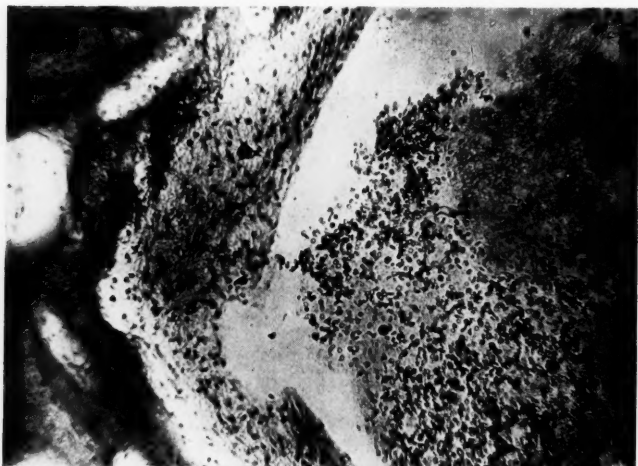


Fig. 6. Hemorrhage in the scala vestibuli, basilar turn with cellular exudate. (Case 6.)

general condition continued poor. Diarrhea and vomiting had stopped. On March 4th, an abscess on the back was incised. Blood culture was negative. On March 5th, both ears began to drain again; the general condition was still poor. There was moderate dehydration and vomiting; the diarrhea had subsided. On March 7th, the ears were worse than when last seen, but there were no definite signs of mastoid involvement on either side. On March 8th, there was a purulent discharge from both ears. On March 9th, the diarrhea was again severe, but there was no vomiting. There was a profuse discharge from both ears. There was a difference of opinion among the consultant otologists regarding the necessity of a mastoid operation. Because of the bronchopneumonia, it was decided to withhold surgery for the present.

Autopsy showed: Acute bilateral media and mastoiditis; diffuse bronchopneumonia; chronic atrophic peritonitis, involving the lower ileum and the peritoneum lower wall.

CASE 6.—J. R., age four months, admitted April 15, 1932. The baby turned a queer color shortly after birth and an injection of the mother's blood was required. There was a persistent loss of weight, and he refused to take the formula well. He gained one pound in the past week on the present formula. The child turned up his eyes and there was a marked photophobia. There was a mucopurulent discharge from both sides of the nose. The hard palate was high and arched. There was a lenticular opacity in the anterior pole of both eyes.

The diagnosis was bilateral congenital glaucoma; bilateral lenticular cataract.

The red blood count was 2,760,000 on admission, 45 per cent hemoglobin: On April 21st, the temperature was 103. On April 25th, a bilateral myringotomy was performed. On April 27th, following the myringotomy the temperature stayed flat; the child had lost weight and was not retaining its feedings. There were still three foul-smelling, semi-solid stools a day. His general condition was poor. The child died on April 29, 1932. The clinical diagnosis was bronchopneumonia; marasmus; congenital glaucoma.

The postmortem findings were: Old cerebral basal hemorrhage, organizing (probably birth injury); diffuse bronchopneumonia; pulmonary edema and congestion; bilateral otitis media and mastoiditis; hypertrophy of the prostate with partial urinary obstruction resulting in hypertrophy of the bladder, ureter and hydro-nephrosis; uric acid, renal infarcts; localized periportal fibrosis of the liver; congenital defects of the eyes. Wassermann was negative; red count persistently low.

CASE 7.—D. P., age six months, was admitted March 24, 1932. On Monday, March 21, 1932, the child developed a severe head cold and a fever. On Tuesday, March 27th, the mother noticed a peculiar staring expression in the child's eyes and foaming at the mouth, associated with general clonic convulsions. A diagnosis of meningitis was made. The parents were told that the case was hopeless, so they removed the baby from the hospital. He was admitted to the Post-Graduate Hospital March 24, 1932. The spinal tap on March 24th showed slightly cloudy, bloody fluid with a cell count of 210. On March 25th, the child had severe, generalized convulsions, which were predominately right-sided. Culture of the spinal fluid showed pneumococci. The ears were examined today, and both drums were slightly bulging. Immediate operation was advised, although the chances of recovery were slight. A left simple mastoidectomy was performed and the brain was explored for an abscess, but none was found. The child died March 25th.



Fig. 7. Showing a dehiscence extending from the hypotympanic cells through the body of the bone to the dura. (Case 7.)



Fig. 8. Pus in the niche of the round window. Serous exudate with cells on the inner surface of the secondary tympanic membrane. Thickening of the mucous membrane with infiltration. (Case 8.)



The clinical diagnosis was acute pneumococcus meningitis and mastoiditis, left.

The postmortem diagnosis was diffuse fibrinopurulent meningitis; bronchopneumonia; pneumococcus septicemia; cloudy swelling of parenchymatous organs.

Note: The blood culture revealed pneumococcus, type undetermined.

CASE 8.—N. G., age 5 months, was admitted April 16, 1932.

The patient had an operation two weeks ago for harelip. The patient had vomited continually since Sunday, April 9th, whenever food was given; it was projectile in character and was equal to the amount of the feeding. The vomiting had occurred only since the operation; previously the baby was doing very well and had gained in weight.

The impression was dehydration and postoperative harelip; acidosis.

On April 21, 1932, a furuncle was noticed on the back of the head. It was incised and pus was obtained. The feeding was by Gavage. On April 27th the child was still vomiting, but the condition was slightly improved. The right drum was bulging; the left drum was negative. For the past two days the child had had a shrill cry. No meningeal signs were present. On May 2nd a bilateral myringotomy was performed with pus resulting. On May 3rd, a bilateral mastoidectomy was recommended. On May 4th, a transfusion of 110 cc. of blood was given. The temperature elevated, and both ears discharged a small amount. The patient retained most of his feeding following the myringotomy; vomiting practically ceased and the stools improved slightly. The child died three weeks from the date of admission.

The clinical diagnosis was bronchopneumonia; dehydration and acidosis; bilateral O. M. P. A.; harelip, postoperative.

The autopsy findings were multiple localized pulmonary abscesses in the lung periphery with necrotizing pleurisy; fatty metamorphosis and cloudy degeneration of the liver and kidneys.

#### SUMMARY.

1. Three routes for meningeal invasion from the middle ear are demonstrated in the pathologic preparations, i. e., directly through the temporal bone, through the bony canals housing the nerves and by traumatic dislocation of the stapes.
2. The transmission of infection through the temporal bone may be facilitated when an acute infection is added to a chronic degenerative process.
3. The membrana tympani is a comparatively resistant structure and peculiarly so following the changes produced by prolonged inflammation.
4. Resolution of the membrana tympani may be present concurrently with a destructive process in the mastoid antrum of infants.
5. No convincing evidence was found indicating that the infected mastoid process acted as a focus for general infections.
6. The classic symptoms of a parenteral infection in infants occurring concurrently with mastoiditis do improve following mas-



toideotomy. The earlier the operation is performed after gastrointestinal symptoms are manifest, the lower mortality.

7. The premature closure of the mastoid incision may cause a recurrence of gastro-intestinal symptoms.

8. The pathology found in the temporal bones we have examined is not a terminal pathology but gives histologic evidence of chronicity.

9. The observation of intralabyrinthine pathology suggests the theory that the vomiting observed, particularly in meningitis, may be the result of labyrinthine irritation.

Dr. Frederick A. Hemsath is the pathologist associated with me in this work, and I am indebted to him for the preparation of the specimens, microphotographs, lantern slides and pathologic opinions.

The fund allotted to me by the Scientific Committee of the American Otological Society permitted the preparation of our histologic specimens.

A complete bibliography may be found by consulting an article published in the New York State Journal of Medicine on January 1, 1930. The title was "Ear Infections in Babies," by Marvin F. Jones and Joseph M. Gerstley.

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#### LXIV.

### THE PARAPHARYNGEAL SPACE: AN ANATOMICAL AND CLINICAL STUDY.\*

COLBY HALL, M. D.,

LOS ANGELES.

Within recent years much consideration has been given deep cervical suppuration, particularly that occurring in the so-called pharyngomaxillary or parapharyngeal space. It is gratifying to see more frequent reports of parapharyngeal infection appearing in the literature, for it is through the study of these reports that we shall more readily recognize disease in this region and be able to treat it in such a manner as to decrease perceptibly the incidence of serious and often fatal complications. For this reason the following detailed anatomic and clinical study of this space has been made.

#### ANATOMY.

A general knowledge of the deep cervical fascia is essential in order to understand more clearly the etiology, symptoms, complications and therapy of infection in the various regions of the neck.

Before encountering the deep fascia one must cut through the skin, superficial fascia (which is not a distinguishable layer), subcutaneous tissue, and anteriorly, the platysma. The space lying superficial to the deep fascia contains superficial glands, nerves and vessels, the most important of which is the external jugular vein.

All of the important structures of the neck are contained within the bounds of one great cervical sheath, which extends from the base of the skull above to the upper end of the thorax below. (Fig. 1.) The superior attachments are the external occipital protuberance, the superior curved line of the occipital bone, the mastoid portion of temporal bone, the zygoma and the outer edge of the mandible. Inferiorly, the attachments are the seventh cervical spine, the spine of the scapula, the acromium, the clavicle and the upper end of the sternum. Posteriorly it is attached to the ligamentum nuchae, between the external occipital protuberance and the seventh cervical spine. Anteriorly the two sides are continuous and are attached in

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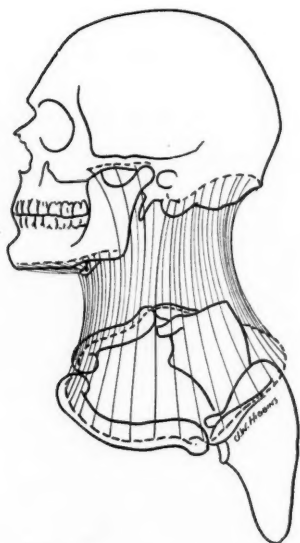


Fig. 1. The Great Carotid Sheath. The broken lines indicate the points of attachment, and the solid black lines connecting them indicate the fascia.

the midline to the symphysis of the mandible and to the body of the hyoid bone. This great cervical sheath is usually referred to as the superficial layer of the deep fascia. Medial extensions of this layer give each structure its own individual fascial envelope. From a practical standpoint there are two deeper divisions of the fascia colli, (1) the visceral fascia or sheath, and (2) the prevertebral fascia.

Figure 2a is a diagrammatic cross section showing these layers of the deep fascia. Within the visceral sheath lie the respiratory, vocal and deglutitory organs in the neck, and in addition, the thyroid and parathyroid glands. The visceral sheath is incomplete anteriorly in the nasal and oral regions and extends from the base of the skull above into the thorax below. It forms the external fascial covering for the pharyngeal constrictors and the buccinator (buccopharyngeal fascia). Anteriorly it is attached to the mylohyoid line of the mandible and to the hyoid bone. Below the hyoid it descends behind the hyoid depressors and in front of the larynx and trachea (pretracheal fascia). From an area between the anterior portion of the trapezius and the posterior portion of the sternomastoid muscle of one side there is sent an extension of deep fascia across the midline to meet a corresponding extension from the opposite side. Where this extension becomes quite thickened and passes in front of the bodies of the

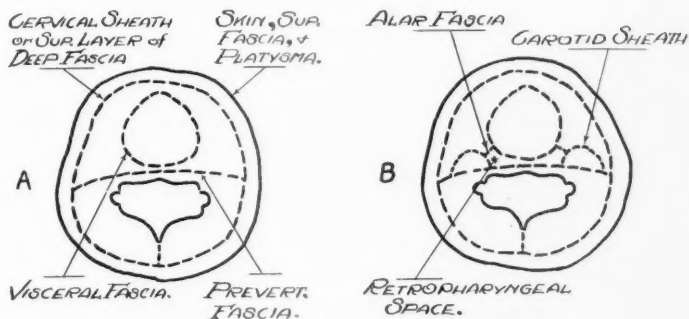


Fig. 2.

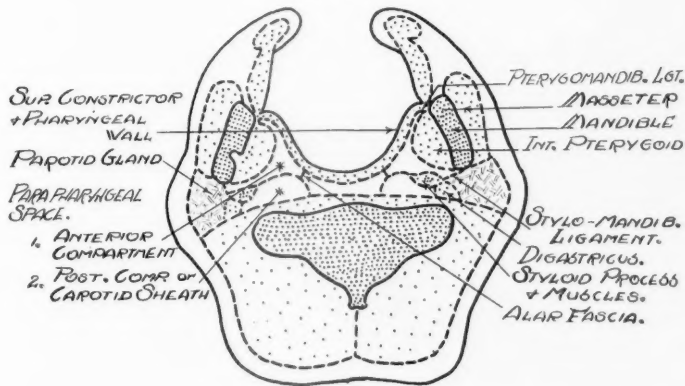


Fig. 3.

Fig. 2. Diagrammatic cross-section of the neck. A—Showing the primary fascial layers. B—The carotid sheath and the alar fascia have been added.

Fig. 3. Diagrammatic cross-section at the level of the mandible. This shows the formation of the parapharyngeal space.

cervical vertebrae, the scalenus anticus and longus colli muscles and the phrenic and sympathetic nerves it becomes known as the pre-vertebral fascia. Superiorly it extends to the base of the skull. Inferiorly in the midline it descends behind the gullet into the thorax, the space lying between it and visceral fascia being known as the retropharyngeal space. At the sides it forms the posterior wall of the carotid sheath (Fig. 2b), covers the scalene muscles and passes downward in front of the subclavian vessels and the brachial plexus until it disappears beneath the clavicle. Here it splits to form the sheath for the axillary vessels.

Figure 2b shows the carotid sheath added to the diagram and also demonstrates a small but very important fascial element—the

alar fascia. This is a fascial expansion which forms a continuation between the carotid sheath and the visceral fascia. Its importance lies in the fact that it completes the anterolateral aspect of the retropharyngeal space throughout the entire length of the neck and separates this space from the lateral pharyngeal regions. This layer is of utmost importance in Dean's<sup>1</sup> external approach to a retropharyngeal abscess.

Figure 3 is a diagrammatic representation of the region of the mandible. The necessary structures have been added to the diagram to show the formation of the parapharyngeal space and its two compartments.

If we now refer to Beck's<sup>2</sup> recent work, we find that the superficial layer of the deep fascia (the great cervical sheath) sends in a deep extension to encapsulate the submaxillary gland, and also another extension, the deep parotid fascia, which completes the capsule of the parotid gland, except superiorly, as will be shown later.

Thus there are, from a clinical standpoint, four definite deep cervical spaces.

- (1) Submaxillary space.
- (2) Parotid space.
- (3) Retropharyngeal space.
- (4) Parapharyngeal space.
  - (a) Anterior compartment.
  - (b) Posterior compartment (carotid sheath).

The peritonsillar space should be mentioned here also because of its close relationship to the parapharyngeal space, being separated from it merely by the superior pharyngeal constrictor muscle and its fascial covering.

Before continuing to the anatomy of the parapharyngeal space it may be well to outline and reiterate the important points concerning the deep cervical fascia.

#### *A—Practical Divisions.*

1. Great cervical sheath.
2. Visceral sheath.
3. Prevertebral fascia.
4. Alar fascia.

#### *B—Distribution.*

Every deep structure contained in the neck possesses its own individual fascial covering which it receives from the deep fascia.

*C—Characteristics.*

1. In certain areas the fascia becomes very thickened and prominent—the carotid sheath, for example. In other areas it becomes so thickened as to form so-called ligaments, such as the stylomandibular and the pterygomandibular.
2. In some regions of the neck there are reduplications or reflections of the fascial covering of one structure so that it becomes directly continuous with the fascial covering of an adjacent structure. The alar fascia is an example of this. This plays a most important part in the formation of the anterior compartment of the parapharyngeal space.

## PRACTICAL ANATOMY OF THE PARAPHARYNGEAL SPACE.

This anatomic region has been described as the pharyngomaxillary, the peripharyngeal, the lateral pharyngeal and as the parapharyngeal space. The term "parapharyngeal space" seems the most logical and will be used throughout the paper.

The parapharyngeal space, as has been described by others, is composed of two compartments, an anterior or pre-styloid, and a posterior or retrostyloid, as shown in Fig. 3. In all dissections by the author these have proven to be separate and distinct compartments, although closely related anatomically and pathologically.

The posterior compartment is the carotid sheath and its contents; it extends from the base of the skull to the upper end of the thorax.

The anterior compartment is normally a potential space and becomes actual only when it becomes the seat of disease. It contains a small amount of connective tissue and usually a few lymph glands; it extends from the base of the skull above to the angle of the jaw below. (Fig. 4.)

Figure 5 is a cross section at the level of the faucial tonsil and shows the boundaries of the anterior compartment. Anteromedially is found the buccopharyngeal (visceral) fascia covering the superior constrictor; this becomes thickened anteriorly at the pterygomandibular ligament and is reflected onto the fascial covering of the internal pterygoid muscle which forms the anterolateral boundary. This in turn is continuous with the stylomandibular ligament (medial parotid fascia), the posterolateral boundary. Posteriorly, we find the

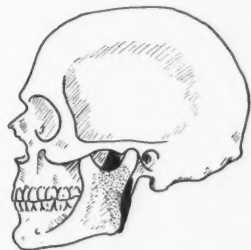


Fig. 4. The solid black and stippled area designates the extent of the distended anterior compartment.





fascial covering of the styloid and its muscles, the anterior wall of the carotid sheath, and posteromedially the alar fascia (stylopharyngeal aponeurosis of Zuckerkandl.<sup>10</sup>) In all dissections this is found to separate completely the anterior compartment and the retropharyngeal space. All of these fascias are continuous and form a complete fascia-lined space, except superiorly where the medial parotid layer is absent. In Figure 5 the carotid is shown in closer relationship to the tonsil than is usual, but the proximity of the pharyngeal wall to the anterior compartment is clearly emphasized.

Figure 6 is a section at the level of the inferior turbinate. Here the anterior compartment is shown distended as in 5a. The compartment measured 4 cm. in depth from this level to its termination at the angle of the jaw.

Inferiorly, the anterior compartment ends blindly in a cul-de-sac at the angle of the jaw. This is brought about by the reflection and reduplication of fascia mentioned earlier in this paper. The fascial covering of the styloid process and its musculature become reflected onto the internal pterygoid fascia; medially, the alar, the buccopharyngeal and the stylomuscular fascia become continuous. The fascial fold (Fig. 7), which is formed at the angle of the jaw, is the inferior limit of the anterior compartment and is the point of entrance to this compartment. Laterally the stylomandibular ligament (which has been divided in Fig. 7) becomes continuous with this fold, completing the compartment. Medially, above the level of the superior constrictor, the buccopharyngeal fascia becomes continuous with the pharyngeal aponeurosis and the covering of the levator and tensor palati muscles.

In Figure 8 is shown the distended end of the anterior compartment as viewed from below. This head was sectioned at the level of the inferior turbinate and the space was distended from above. This blind ending of the anterior compartment was found repeatedly in a large number of cadavers by Dr. Bartosh of the Anatomy Department of the University of Southern California. A most unusual and interesting demonstration of the anterior compartment in the living person was made by Weidlein.<sup>11</sup> He reported a latent parapharyngeal abscess and displayed his X-rays after the instillation of 15 cc. of lipiodol into the cavity. His paper is most instructive. The relationship of the external maxillary artery and the hypoglossal nerve is clearly shown in Figure 8, the facial vein having been cut and the submaxillary gland lifted somewhat out of its bed.

In Figure 9 are shown the various fascias which come into play in forming the more medial wall of the anterior compartment. The



Fig. 7.

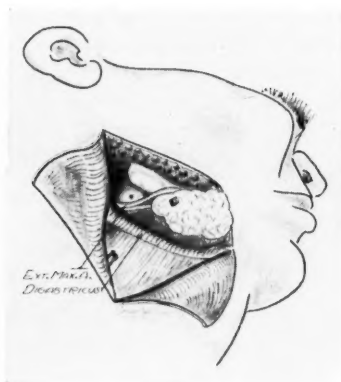


Fig. 8.



Fig. 9.



Fig. 10.

Fig. 7. The parotid gland and the stylomandibular ligament have been divided and retracted to show the lateral aspect of the parapharyngeal space. This is also from a dissection.

Fig. 8. This dissection shows the distended blind end of the anterior compartment as indicated by the asterisk. The head has been sectioned at the level of the inferior turbinate and the compartment distended from above. The facial vein has been cut and the submaxillary gland lifted somewhat from its bed. The external maxillary artery is shown in its close relationship to the anterior compartment. The hypoglossal nerve has been dissected out and is shown beneath the artery. Fibers of the mylohyoid muscle are seen through the fascia beneath the gland.

Fig. 9. Dissection showing the more medial portion of the parapharyngeal space. The alar fascia has been removed superiorly to reveal the fascial relations and the relations of the retropharyngeal and parapharyngeal spaces. The points of the hemostat are seen entering the space from within the throat.

Fig. 10. Dissection showing the simple external approach. Tissues including the great cervical sheath have been cut through and the hemostat is shown entering the anterior compartment behind the angle of the jaw.

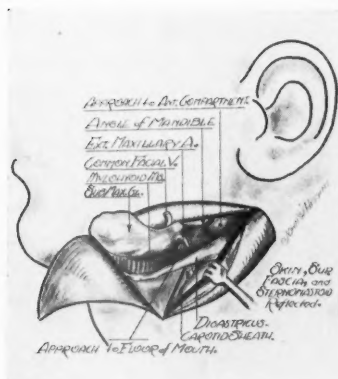


Fig. 11. Dissection showing the approaches to the various fascial spaces as offered by the Mosher Operation.

importance of the styloid process as a surgical landmark is clearly shown. Posterior to this and more or less protected by it and its musculature is the carotid sheath.

The close relationship existing between the parapharyngeal, the parotid and the submaxillary spaces has been shown very clearly, by Beck.<sup>2</sup>

#### ETIOLOGY.

Etiologically, there are three types of parapharyngeal infection. First, there is the type which follows tonsil surgery and which is almost exclusively preceded by local anesthesia. Second, there is the type in which there is an extension from a neighboring compartment. Third, there is the type in which the parapharyngeal space is the seat of the initial invasion of the deep fascia, and which is not preceded by throat surgery. According to A. L. Beck,<sup>2</sup> the etiology here is most commonly in the tonsil or pharynx; although he states that infection in the nose, sinuses, adenoid, pharyngeal lymph nodes, cervical vertebrae, mastoid and petrous bone are all possible sources.

I am in accord with Christerson<sup>1</sup> that parapharyngeal abscess should not be included under retropharyngeal disease, but do not agree that it always follows tonsillectomy.

#### PATHOLOGY.

As early as 1910 Dean<sup>3</sup> reviewed the literature on deep cervical infections resulting from tonsil operations and reported three cases of his own. Shapiro,<sup>4</sup> in 1930, published a very complete treatise on

post-tonsillectomic cervical infection and reported 110 cases, thirty being in his own practice. He says that there are two forms of cervical involvement: (1) The phlegmonous type: This is the most common and comprised 94 per cent of the 110 cases. Here the signs of local inflammation are predominant. There is a cellulitis of the loose tissue in the anterior compartment, to which it probably remains confined in uncomplicated cases. Abscess formation takes place in the majority of cases. (2) The vascular type: This is rare and comprised the remaining 6 per cent of Shapiro's cases. The prognosis is bad and the local signs are usually absent. The symptoms are those of septicemia, thrombosis or embolism without preceding or accompanying signs of cervical phlegmon.

There are three possible routes by which the parapharyngeal space may become infected: (1) Direct implantation, caused by the penetration of an injection needle or some other instrument through the fasciomuscular wall into the space; (2) the vascular route, in which there may be an extension of an endophlebitis or thrombosis of the parent vessels, as demonstrated by Waldapfel;<sup>7</sup> and (3) the lymphatic route; this may occur through suppuration of the deep cervical nodes or, as Shapiro says, there may be a lymphangitis producing a phlegmonous infection without any suppuration.

Those cases occurring not postoperatively and in which the anterior compartment is primarily invaded following some upper respiratory infection are probably all caused via the lymphatic route. In these cases the cellulitis and suppuration transform the potential space into an actual one. Unless the pus is evacuated and free drainage instituted any of the following complications listed by Shapiro<sup>6</sup> may ensue:

1. Mediastinitis.
2. Jugular thrombosis.
3. Edema of the larynx.
4. Ludwig's angina.
5. Hemorrhage of the large vessels.
6. Osteomyelitis of the mandible.
7. Pneumonia.
8. Erysipelas.
9. Vagus involvement (sudden death)?
10. Meningitis.
11. Parotid abscess.

Mention should now be made of vessel pathology. Lifschutz<sup>8</sup> states that the blood vessels are usually the last to yield in the case of

an abscess or sloughing infection in the deep cervical space. He names the following four factors in this protection afforded by nature: (1) Perivascular lymphatics and nourishing vessels; (2) fascial sheaths, such as the carotid sheath; (3) specific reaction to infection tending to prevent sudden rupture of (a) a vein (pathologic sequence of periphlebitis, phlebitis and thrombosis), and (b) an artery in which the pulse pressure is too great and the stream too rapid to allow easy clotting. However, the force acting against a weakened spot tends to produce an aneurysm or sacculatation; (4) thick vascular coats and the probable phagocytic quality of the endothelium.

In the case of either the artery or the vein the pathologic process is relatively slow and of such character as to prevent sudden rupture and severe hemorrhage.

#### SYMPTOMATOLOGY.

There are four cardinal signs of involvement in the anterior parapharyngeal compartment. They are, in the order of importance:

1. Inability to open the mouth widely.
2. Induration about the angle of the jaw.
3. Fever which may be septic in character.
4. Medial bulging of the pharyngeal wall.

These symptoms follow some recent throat operation or an upper respiratory infection either existing with the symptoms or having been present only a few weeks prior to their onset.

Each of these symptoms has a clear anatomic reason for its existence. I agree with Iglauer<sup>9</sup> that trismus is the most important sign and results from the close relationship of the infection to the internal and external pterygoids. It is the same as that seen in a case of peritonsillar abscess; however, in parapharyngeal abscess there is also induration at the jaw angle.

If we remember that the compartment ends at the level of the angle of the jaw it becomes clear why induration and swelling should begin in this region. As the infection continues the area of induration becomes wider. If we recall that at its most superficial part in the neck, at the angle of the jaw, the anterior compartment is separated from the surface by two thick complete fascial layers, the superficial layer of the deep, and the blind end of the space or fascial fold, we can readily see why fluctuation occurs late, if at all, and therefore should not be awaited.

Any infection, pent up such as infection is in this region, will cause fever. Another point to be considered is the close relationship to the great vessels.

Pharyngeal bulging is easily explained by the thin fasciomuscular wall between the pharynx and the compartment. It must be added, however, that the trismus may be so marked as to preclude a thorough pharyngeal examination. This symptom may not be marked, as noted in one case described later.

Points in differential diagnosis will be discussed in report of cases.

#### TREATMENT.

The treatment of parapharyngeal infection is surgical. When the above symptoms present themselves early surgical drainage is imperative. To await fluctuation in the presence of these symptoms is to await complications. Mosher,<sup>10</sup> in 1920, emphasized this and stated that infection here should be considered as a perijugular abscess comparable to perisinus abscess in the mastoid and should be treated accordingly. His treatise should be studied very carefully. There are three surgical approaches to the parapharyngeal space—one pharyngeal and two external.

*Pharyngeal Approach.*—A curved hemostat is thrust through the pharyngeal wall, superior constrictor and buccopharyngeal fascia where the bulging is greatest, much the same as in a peritonsillar abscess. Figure 9 shows the tip of the hemostat protruding into the anterior compartment. This approach is probably of value only in infection localized to the anterior compartment.

*External Operation.*—Two approaches. Shapiro<sup>6</sup> lists the following indications for external drainage:

1. If the abscess points externally only—that is, when there is no bulging of the tonsil fossa to be seen and the symptoms of sepsis are increasing.
2. If no drainage has taken place within from twenty-four to forty-eight hours after intra-oral operation.
3. If the external swelling and the symptoms of absorption progress, even though the abscess is draining through the pharyngeal incision.
4. Upon imminent approach or the actual presence of one or more of the complications mentioned.

Personally I believe an external operation is preferable in practically every case.

*Simple Approach* (Figure 10).—If the case is operated upon early when infection is limited to the anterior compartment and before any complications have set in this will suffice. An incision is made parallel to and below the mandible, extending from a point

between the angle of the jaw and the mastoid tip posteriorly to a point about half way between the symphysis menti and the angle of the jaw anteriorly. The skin, the superficial fascia and the platysma are cut through, exposing the superficial layer of the deep fascia or great cervical sheath. This is incised and retracted and at a point just behind the angle of the jaw (Figs. 8, 10, 11) the finger or a hemostat is plunged into the anterior compartment. Upon entering the compartment one palpates the styloid process posteriorly and the stylomandibular ligament laterally. If the parotid or submaxillary spaces are involved they may be entered by this route. The individual may perhaps wish to make a different incision than the one described above, but he will find the same relations existing in the region of the angle of the mandible. The induration and congestion of the tissues make the operation much less simple than upon the cadaver, but if one keeps the anatomy of this region and the extent of the anterior compartment clearly in mind he should encounter little difficulty.

*Mosher's Approach* (Figure 11.)—This was described by Mosher,<sup>11</sup> in 1929, and is indicated in all cases where complications have occurred and in which the carotid sheath and its contents are to be exposed. A study of Figure 11 will reveal the possibilities of this method. To appreciate the more detailed surgical anatomy of this approach the original article should be carefully studied.

#### REPORT OF CASES.

CASE 1.—*Abscess superficial to the great cervical sheath.*—M. L., a white girl, aged 3 years, was admitted to the Children's Hospital on May 9, 1933, with a swelling of the right side of the neck. She had an otitis media on the right and had had a cold for one month before admission. The temperature was 101° F. There was an extremely hard swelling at the angle of the right jaw, extending anteriorly over the region of the submaxillary gland. The tonsils were hypertrophic, cryptic, red, and they met in the midline. On May 12th the submaxillary region was very hard, swollen and tender; the area of swelling had extended up and over the ramus of the mandible. Hot, moist applications were continued to the affected area. By May 16th a mass, the size of a small lemon, had definitely localized in the submaxillary area and it was quite fluctuant. The temperature during the week in the hospital remained between 100 and 101 degrees F. rectally, and there was no trismus, pharyngeal bulging or severe general symptoms.

On May 17th an incision was made over the swelling and a superficial abscess cavity, containing about an ounce of pus, was found. The child made an uneventful recovery.

This child was treated along conservative lines and kept under observation. At no time did she appear septic, nor did she present either trismus or pharyngeal bulging or protrusion, so we felt per-



fectly safe in awaiting either fluctuation or subsidence of the mass. Operation revealed an abscess lying between the skin and the superficial layer of the deep fascia.

CASE 2.—*Abscess deep to the great cervical sheath and to the sternomastoid muscle.*—F. G., a white boy, 12 years of age, was admitted to the Children's Hospital on May 19, 1933. He had a swelling at the angle of the left jaw of twelve days' duration, and dysphagia of three days' duration. Onset of the present illness was an acute cold three weeks prior to admission. The temperature was 100° F. He had a bilateral otitis media. The left tonsil was pushed to the midline, but there was neither redness nor induration of the peritonsillar tissues. Below the angle of the left jaw there was a swelling about the size of a large lemon, not definitely circumscribed, nonfluctuant and quite tender. There was no trismus or swelling in the floor of the mouth. He had a leukocytosis of 14,000 and a red cell count of 4,540,000. On the following day he appeared quite toxic and the temperature had risen to 102.4° F. Surgery was decided upon.

The anterior compartment of the parapharyngeal space was explored with the finger, but no pus or cellulitis was found. An abscess was found deep to the sternomastoid muscle. The tonsil was removed because it impeded respiration. The temperature rapidly returned to normal. Child was discharged from the hospital on May 31st with the wound healed and the general condition good.

In this case the pushing medially of the tonsil and the swelling just below the angle of the jaw made exploration of the space seem imperative. However, the important points opposing parapharyngeal involvement were lack of trismus and lack of intense induration about the angle of the jaw. This abscess cavity was deep to superficial layer of the deep fascia.

CASE 3.—*Primary syphilitic reaction with parapharyngeal symptoms and associated with a suppurative adenitis.*—C. B., a white male, aged 22, came to the office on June 12, 1933, complaining of a painful swelling in the right side of the neck of three weeks' duration. He had had a cold and a sore throat several weeks previously, but did not complain of a sore throat when seen in the office. The temperature was 100.5° F. The tonsils were slightly reddened without any ulceration or membrane. In the right side of the neck, about one-half way down, the sternomastoid muscle was a mass about the size of a small orange, rounded, firm and very tender. There was no edema or displacement of the pharyngeal wall and the mandible was freely mobile without pain. On June 13th an abscess, deep to the sternomastoid, was opened and drained, and the patient was greatly relieved. On June 15th he returned to the office with the neck again stiff and swollen and with induration limited to the area of the wound. There was extreme trismus, but without induration behind the angle of the jaw, although this region was very tender. The right tonsil now presented a large, ragged, deep ulceration at the upper pole. The edges of the ulcer were firm and there was a dirty gray membrane covering the floor of the ulcer, the disturbance of which caused slight bleeding. Laboratory findings on June 16th: W. B. C., 14,000; 90 per cent polys. Culture negative for diphtheria. Smears from ulcer showed no acid-fast or spirochetes, but did show gram positive diplococci and gram negative fusiform bacilli.



On June 17th the neck was re-opened and small amount of pus recovered. Drain was again left in the wound. He was discharged the next day feeling greatly relieved. The tonsil lesion was subsiding. The patient did not return until June 29, 1934. During this interval the blood Wassermann had been reported four plus and checked. On this date the neck was entirely healed, there was no trismus and the tonsillar lesion had disappeared. However, there was a diffuse papillo-syphilide on the body which had appeared the previous day. The boy was referred to Dr. Stanley O. Chambers.

This case is reported because it emphasizes the importance of thorough diagnostic investigation of pharyngeal infections, as noted by Costen.<sup>12</sup> In this boy there was a cervical adenitis which probably was secondary to the upper respiratory infection occurring previous to his consulting a physician. Dr. Chambers considered the lesion on the right tonsil as the primary sore or chancre. The marked trismus here was due to a specific reaction of the tissues of the peritonsillar, and perhaps the parapharyngeal, space adjoining the acute luetic tonsil. This was proven, I believe, by the prompt subsidence of symptoms about the pharynx, absence of sepsis and satisfactory course of the suppurative adenitis.

CASE 4.—*Parapharyngeal abscess following sinusitis and orbital abscess.*—J. S. B., a white boy, aged 6½ years, was sent into the Children's Hospital on May 22, 1933, with a proptosis of the right eye, and a diagnosis of probable orbital abscess. Onset of illness was on May 15, 1933, with a swelling of both eyes, slight swelling in the left neck but with no increase in temperature. The edema of the left eye had receded by May 19th, but the painless swelling of the right eye had increased and was associated with a nasal discharge. Past history included rather frequent colds. Patient had had a tonsillectomy in 1932. The temperature was 99.8° F. upon admission. There was an edema and redness of the upper and lower lids on the right, and a moderate proptosis. The pupils were dilated and equal. The right nostril was filled with a mucopurulent discharge and there was such intense swelling of the membrane on the right side of the nose that there was no breathing space. No cervical adenopathy noted. Conservative measures were instituted and the boy showed some improvement. However, on May 26th, there was such an intense orbital swelling, and, in spite of attempted shrinkage, the right nasal space was so completely occluded it was decided that surgical interference would be necessary. There was a proptosis of the globe with slight lateral displacement. The pupillary and extraocular muscles reacted normally, and vision was normal to gross tests. On the right side of the nose the inferior turbinate could be seen completely obstructing the space and it was covered with yellow pus.

An external ethmoidal drainage was performed on May 27th, and an inferior meatal window was made in the right antrum. A perforation in the lamina papyracea was found and there was some yellow pus between the bone and periosteum. A large opening was made into the nose and a through and through rubber drain inserted.

There was an occasional flare-up of the naso-orbital condition, but on June 12th the external wound was healed and the nose was clear. The temperature, which had risen to 102° F. postoperatively, had remained normal since June 3rd. Beginning on June 16th and occurring each day thereafter was an increase of the

temperature to between 101 and 102 degrees F. This occurred each afternoon at approximately four o'clock. On June 22nd a swelling appeared on the right side of the neck. On June 23rd this had increased in size and the temperature had risen to 103°.

On June 24th there was an intense cellulitis of the right neck, the boardlike induration extending from the region of the mandible almost to the clavicle; the overlying skin was tense, glistening, extremely tender but not red. Associated with the above was a pronounced trismus, the child being barely able to open his mouth. There was no involvement of the oral floor and no medial displacement of the lateral pharyngeal wall could be seen. There was a small amount of lymphoid tissue in each tonsil fossa, but these were not inflamed.

On June 24th the neck was opened. The simple incision along the mandible was made and the superficial layer of the deep fascia cut through, the submaxillary capsule was located and found to be moderately swollen. The anterior compartment was entered with the finger; the styloid, stylomandibular ligament, and the base of the skull were palpated. A dram of frank yellow pus was recovered under tension and a rubber drain was inserted. Culture from the neck revealed hemolytic streptococcus.

Hemolytic streptococcus was cultured from both the ethmoid and from the neck abscess.

On June 29th the swelling had subsided, the general condition was good, and the temperature had returned to normal. The patient was last seen on October 26, 1933, when he visited the outpatient clinic. He was in very good physical condition.

This lengthy report is given because of the unusual nature of the case, being the only parapharyngeal infection I have seen reported following sinus infection or orbital abscess. Following the ethmoid drainage the patient's temperature became normal in a week; within two weeks the wound and the nose were clear.

The date of onset of the neck infection, I believe, should be considered as June 16th, when, after having been normal for two weeks, the temperature began its daily fluctuation by rising to 102°. It should be recalled that the neck swelling did not occur until six days later. June 16th was just two months after the onset of the original illness and three weeks after ethmoid drainage. The exact importance of these time intervals I do not know; but it is a very significant fact that the cultures from the orbit and from the parapharyngeal space were identical: hemolytic streptococcus.

The combination of the following signs, (1) intense boardlike induration extending from the submaxillary region almost to the clavicle, (2) septic temperature, and (3) extreme fixation of the jaw pointed to parapharyngeal infection.

As reported, within forty-eight hours after the first appearance of the neck swelling the space was opened and pus recovered. This

was followed by prompt subsidence of symptoms. This shows clearly the folly of awaiting fluctuation when the infection is in the anterior compartment. To have procrastinated would probably have meant the onset of complications.

*CASE 5.—Erosion of the internal carotid artery into the parapharyngeal space.*

—G. S., a white boy, aged 2½ years, was admitted to the Children's Hospital on March 31, 1934. There was a history of sore throat of six days' duration, swelling on the left side of the neck, and dysphagia for four days. The temperature upon admission was 99° F. The left tonsil was very red and was pushed beyond the midline by a very pronounced peritonsillar inflammatory swelling. There was no evidence of retropharyngeal involvement. There was a moderate trismus and a slight induration beneath the angle of the left jaw. There was also an enlargement of the glands along the upper portion of the sternomastoid; these were quite firm and tender. W. B. C., 41,000; 90 per cent polys; R. B. C., 4,470,000; 86 per cent hemoglobin. The mucous membrane was incised at the usual site for a peritonsillar abscess and artery forceps were inserted. The tips of the forceps were barely spread when there discharged some dark reddish black clots from the wound. These were then followed by larger and brighter clots and by the loss of about one-half an emesis basin of blood. The patient was put back to bed and he looked quite pale and restless. Shortly afterwards his color returned and he seemed better. At 4 p. m. the temperature had risen to 103°; the pulse was rapid but strong. He was given some sodium amytal rectally. However he continued to spit up dark clots from time to time. At 1:00 a. m., on April 1st, he had a small hemorrhage of red blood and became quite restless again. At 1:50 a. m. he died very suddenly without any struggle or hemorrhage.

At autopsy Dr. Hyland, the pathologist, found a dilated pulmonic orifice; acute hyperplasia of the spleen; cloudy swelling of the heart, liver and the kidneys; and the lungs and the heart appeared very pale. Permission to open the neck was not given so we were forced to explore from below. Dr. Hyland reported an intense cellulitis of the pharyngeal tissues. An attempt was made to force water through the internal jugular but to no avail. When the syringe was attached to the cut end of the common carotid the water went in very readily and escaped through the mouth. A long artery forceps was plunged into the parapharyngeal space from below and a cavity filled with dark clotted blood was entered.

The throat presented a picture of peritonsillar abscess with impaired respiration and was treated accordingly by the attending house officer. From the appearance of the throat, that was the only logical thing to have done.

The leucocyte study immediately eliminated leukemia or agranulocytosis. The absence of hyperpyrexia upon admission was rather puzzling in the face of a polynuclear leukocytosis of 41,000. The later increase, however, showed that an acute process was in progress. The presence of hemorrhage into the cavity was proven by the result of the incision. The question then arose as to its source and as to the correct procedure to follow.

If the process was limited to the area medial to the superior constrictor (peritonsillar space), then it must have been caused by one of the tonsillar arteries which are branches of the external carotid. In this case the tonsil should have been removed and the vessel tied.

If the process was external to the constrictor—in the parapharyngeal space—then the hemorrhagic involvement was from the large neck vessels, the internal carotid or the internal jugular. In this case it would have been contraindicated to operate in the throat until the offending vessel was located and ligated in the neck. The intensity of the process, coupled with a certain degree of trismus and induration behind the angle of the mandible, made erosion of one of the large vessels into the anterior compartment seem most likely.

With the child seemingly improved, it seemed advisable to observe him for a few hours and to keep him at rest. It should be noted that he did not die of sudden hemorrhage or asphyxia. The causes of death may have been (1) sepsis, (2) failure of the right heart, or (3) "vagus death"—whatever that may be.

It was proven that a branch of the common carotid was the cause of the hemorrhagic process, and by exploration postmortem it was shown that the parapharyngeal space was filled with clotted blood. The external carotid itself is too far removed from this space to be given much consideration, and its branches which supply the tonsil seem rather small to cause such an extensive process into the parapharyngeal space. It was suggested that an erosion of the internal carotid would have caused sudden hemorrhage by the continued high pulse pressure being exerted into the cavity. This, however, is not necessarily true, as one can readily understand after reviewing the work of Lifschutz. For a complete study of hemorrhage in similar conditions one should review the extensive report by Salinger and Pearlman.<sup>13</sup> Wishart,<sup>14</sup> in 1923, reported a case of internal carotid erosion secondary to retropharyngeal abscess, and his case died a death similar to the one reported above.

CASE 6.—*Tonsillitis with peritonsillar bulging*.—S. F., a white girl, aged 2 years, was admitted to the Children's Hospital on April 4, 1934. She had had an earache on the right and a sore throat for six days. There had been a swelling in the neck for five days. The temperature was 102° F. There was an acute otitis media on the right. Examination of the throat revealed the right tonsil to be pushed almost to the midline, but the surrounding tissues were not red or indurated. The tonsil itself was red and contained some yellowish cryptic material. Neither trismus nor induration beneath the mandible was present. W. B. C., 27,900; polys 90 per cent; R. B. C., 4,390,00. The child was kept in bed and observed. The temperature returned to normal on the following day and remained down. The throat condition promptly subsided and within a week of admission it was entirely normal with the tonsil back in normal position.

This patient having been admitted within a few days after Case No. 5, it naturally caused no little concern as the high temperature, the blood picture, and the medial tonsil bulging were all similar to the picture presented by Case No. 5. However, there being no induration or marked inflammation in the peritonsillar region, incision of this area was deemed unnecessary. Because of the absence of trismus and induration about the angle of the mandible, observation of the patient was decided upon. As noted above the condition returned to normal very rapidly. The swelling in this case was undoubtedly due to an edema in the peritonsillar region or fossa.

CASE 7.—*Parapharyngeal abscess and thrombophlebitis.*—The following case is not one which I attended professionally, but one which I have watched with interest and with no little concern. The patient is about 38 years of age, white, male. In January, 1934, he had an upper respiratory infection. This cleared up. During the middle of February he noted a tender swelling beneath the left jaw. Teeth, throat, and sinuses checked and found to be all right. About five days after the appearance of this swelling the complete left side of the neck adjacent to the mandible became very swollen and indurated. He was sent to the hospital for observation. Hematology showed evidence of an acute process. He had an intense induration extending from the submaxillary region to behind the angle of the jaw. In addition he presented an intense trismus and a septic temperature hitting a peak of between 103 and 104 degrees each day, followed by a return to subnormal with marked perspiration. This continued for a week. A small superficial incision was made, only blood was recovered. No deep exploration made. For four more days the patient became worse and looked extremely toxic, and still no fluctuation. Upon the patient's request the wound was again explored, this time much deeper and higher, followed by a gush of about two ounces of foul yellow pus. Since that time the patient has improved very slowly, still having an occasional septic period. From time to time the wound has been probed and pus recovered, no sizeable incision having been made. When I last saw him there remained a trismus without pain, had had blood transfusions (number not known) and, although still a sick man, was apparently on the road to recovery after many weeks of disability.

This case appears too clear cut to warrant much discussion. He ran the typical course of a sore throat followed by neck swelling, intense trismus and sepsis which definitely point to phlebitis of the internal jugular. Early intervention, as in Case 4, would possibly have prevented jugular involvement, and, if not, the vein should have been exposed. Mosher stressed this many years ago. Attention should be drawn to the inadequacy of both surgical attempts. The first reached only those tissues external to the space, no attempt being made to enter this fossa at the point designated in Figures 8, 10 and 11. Mosher, in 1929, laid down the principle of a sufficiently large surgical wound—which principle was not adhered to in either operation.

The folly and danger of awaiting fluctuation in this region is again emphasized. Two ounces of fluid pus were present and the neck presented an intense boardlike induration.

The remarkable thing to me is that this man is alive today.

## CONCLUSION.

It is hoped that through this study disease in the parapharyngeal space will be diagnosed and rationally treated at such an early stage that the long convalescences and the high mortality consequent to it and its complications will be markedly decreased.

I wish to express my gratitude to John Higgins of the School of Medicine of the University of Southern California for aiding me greatly in making these dissections and for preparing all of the drawings.

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EFFICIENCY OF ANTROSCOPIC EXAMINATION OF THE  
MAXILLARY SINUS.

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Braun,<sup>1</sup> Spielberg,<sup>2</sup> Trotter,<sup>3</sup> Simon<sup>4</sup> and others have pointed out the advantages of antroscopic examination as compared to transillumination, washing and roentgenograms for diagnosis of maxillary sinusitis. Transillumination is of little diagnostic aid. At best it is only corroborative. Washings are of value only when positive, and are negative in the presence of hyperplastic linings. Roentgenograms help us much, but in addition to the extra expense often result in errors. There is nothing more certain in diagnosis than seeing the lining mucosa in the sinus.

Since that time there has been a slight improvement in reducing the diameter of the antroscope, and making the procedure less difficult, but not more effective. Also, there has been added a so-called fore-oblique instrument, but this helps very little. The essential trouble is, as I have said before,<sup>5</sup> that the type of instrument used at present is at fault. Our nasopharyngoscopes and antroscopes were never designed specially for use in the nose and antrum. They were made for infant cystoscopy, and we have had to adopt these instruments for our needs. The principle used for furnishing light is well suited for use in the bladder, but certainly not in the nose, where space is more limited. The lamp and its housing take from 10 to 13 mm. of the tip of the various instruments; which distance is about the size of most antra in the mediolateral dimension. We need an instrument with an objective lens at the distal tip of the antroscope. Light can be furnished indirectly or by carrier to permit the eye of the instrument to get into the small cavities of the nose.

In order to get a greater length of the antroscope into the antrum some have punctured through the canine fossa. I have used this route instead of that through the inferior meatus in several cases and feel that its disadvantages are much greater than its advantages. The advantage of permitting more of the antroscope to enter the antrum to observe more of the mucosa appears highly desirable on first thought. However, after studying a number of specimens I realized



that this is not so. The following work is a study of forty-two antra, in the cadaver, comparing the results of antroscopic examination by a puncture through the canine fossa to that by a puncture through the inferior meatus. The plan used in this work was as follows:

1. Puncture through the inferior meatus measuring the greatest distance the objective lens could pass into the antrum. At the same time the mucosa was observed and data collected. (Table I).

2. Puncture through the canine fossa with the same procedure and observations.

3. Finally, after these procedures were completed, removal of the lateral wall of the antrum to check on the data observed.

From Table II one sees that the greatest average distance that the objective lens entered the sinus through the inferior meatus in twenty-one right antra was 3.78 mm. The minimum in all of this same group was —2 mm. (i. e., 2 mm. short of entering the sinus) in specimen 21R; the maximum was 10 mm. in specimen 30R and 39R. The greatest average distance traversed in twenty-one left antra was 3.98 mm. with a minimum of —4 mm. in specimen 21L and a maximum of 14 mm. in specimen 22L. The greatest average distance for 42 specimens via this route was 3.88 mm.

The distances passed in the case of punctures through the canine route were as follows: The antroscope passed on the average in twenty-one right sided antra for 15.04 mm., with a minimum of 0 in 34R and a maximum of 28 mm. in 22R. For the twenty-one left sided specimens the average was 14.86 mm., with a minimum of 5 mm. in 29L and a maximum of 23 mm. in 24L. The greatest average canine passage for forty-two antra was 14.95 mm. In the forty-two antra the difference for these two routes is 3.88 mm. for the meatal course and 14.95 mm. for the canine direction, or almost four times as long.

The ability to see the ostium in an antrum is in a rough way an indicator of a satisfactory examination of its mucosa. With the antroscope passed through the inferior meatus one could see the ostium in 66.7 per cent of twenty-one right and 52.38 per cent of twenty-one left antra, or 59.22 per cent of the total forty-two antra. The canine route gave positive results of 85.71 per cent of twenty-one right antra and of 76.19 per cent of twenty-one left antra, or 80.95 per cent of the forty-two antra, as against 59.22 per cent of those through the meatal puncture. In other words, the canine route permitted one to examine the antra more satisfactorily in only 22 per



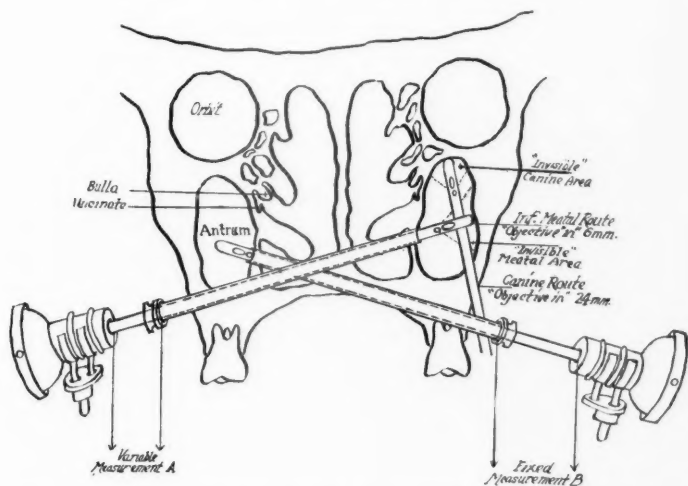


Fig. 1. Diagram to illustrate method for obtaining the distance that the objective lens moved into antrum. The variable measurement A was subtracted from the fixed measurement B (of course on the same antrum) and was done in each route on every antrum.

cent more of the cases, whereas it actually passed 300 per cent greater distance into the sinus. One of the reasons for this ineffectiveness is shown in Fig. 1. There is a so-called "invisible" circular area that cannot be seen by the antroscope through either puncture; that via the canine strikes close to and often includes the ostial area. Furthermore, there is more severe reaction from the canine fossa puncture than from the inferior meatal puncture. The canine route is covered by a much greater amount of highly vascular soft tissue, and the bone to be punctured is thicker, requiring a mallet in practically every case. Puncture through this route is a much more severe procedure, resulting in marked edema, pain, with organization of blood in the malar soft tissue, which we do not see in puncture via the inferior meatus. Because of these severe reactions I have given up puncture via the canine fossa for diagnostic purposes. If the objective lens can enter a few millimeters (and at times even one millimeter) further into the antrum via the inferior meatus one can see the whole interior lining of the sinus. An antroscope with an objective or eye at the distal tip will give more than this needed distance. One can then see the whole antrum, even when the antroscope has barely entered or even when it has not actually entered the cavity. One will be able to see the maxillary ostium and the sphenoid ostium from the middle meatus with little manipulation.

Previously Myerson<sup>6</sup> pointed out that what we call the ostium of the maxillary sinus is usually a canal of varying length. Simon,<sup>5</sup> Jones and Rubin also brought out this fact. In this group of forty-two antra the average distance from the lateral wall of the infundibulum to the internal border of the ostium in the antrum for the normal ostia was 6.17 mm. The minimum for this distance was 1 mm., as in specimens 21R, 32R and 41R; the maximum was 15.5 mm. in specimen 35R. The present group showed a greater average length of the canal than the previous study by 1.5 mm. This can be explained, I think, by the extreme variation that one finds in sinus anatomy. In this particular group there were a few specimens with very long canals of 15.5, 14.5 and 14 mm., etc., while the previous group had only 9 and 7 mm. as the longest canals. These few specimens readily suffice to explain the increased average length. In the earlier work I thought that any opening into the antrum that measured more than 3 mm. in its mediolateral dimension should be called a canal and not an ostium. On this basis over 70 per cent of the normal antral openings in these forty-two specimens are infundibulo-maxillary canals. The accessory openings, on the other hand, are usually not canals. In this group there were eight accessory ostia, of which 87 per cent measured less than 3 mm. in their mediolateral extension. The direction or course of these ostia or canals from the infundibulum into the antrum were divided into two groups. First, those that had any degree of anterior direction, and second, those that had any degree of posterior direction in their courses. The more definite direction was mentioned first and the less definite followed. Of thirty-four openings there were eighteen, or 53 per cent, with an anterior direction; sixteen, or 47 per cent, with a posterior.

Our classification of antral ostia consists of: (1) Normal ostium, any openings from the infundibulum ethmoidale into the sinus; (2) Accessory ostium, any opening outside the infundibulum ethmoidale, in the middle meatus leading into the antrum. Shaeffer and Davis respectively mention duplicate ostia in the floor of the infundibulum. The term "duplicate," when used in this classification, means a complete and distinctly different canal or ostium. The term joining, when used, means two openings contiguous and joined to each other; they have a common opening on one wall and divide to form two separate openings on the other wall. The classification is as follows:

1. Normal canal or ostium, a single opening from the infundibulum ethmoidale into the antrum, as 21R, 22R, etc.
2. Duplicate normal canals or ostia, two distinct and separate openings from the infundibulum with two distinct openings into

the antrum. There were no specimens of this type, though it is not rare.

3. Joining normal canals, with two openings on one side of the canal and one at the other side, as in specimen 41R and 41L, which had two openings into the antrum and one in the infundibulum; the reverse order occurs frequently.

4. Joining normal and accessory canals, composed of a normal canal and an accessory canal which join to have a common opening into the antrum, as in specimen 35R and 39R. This is a common type.

5. Accessory ostia or canals, single openings from the middle meatus and outside the infundibular gutter into the antrum, as specimen 21L, 35L, etc.

6. Duplicate accessory ostia with two distinct openings in the middle meatus outside the infundibulum, as in specimen 31R.

7. Joining accessory canals, two openings in the middle meatus outside the infundibulum or on antral wall uniting to form one opening on other side. This is a rare type and there were none in this group.

Two or more of the above types of opening may occur in the same specimen, as in 35R. I have often felt that the reason we do not recognize more accessory ostia and other types of opening in patients on nasopharyngoscopic examination of the nasal fossa is due to our not having an antroscope with an objective at the tip of the instrument. I hope in the near future I will be able to present such an instrument.

#### CONCLUSION.

1. The slight advantage obtained by puncture through the canine route as against the inferior meatal route in antroscopic diagnosis is worth little, compared to the more severe reaction after puncture via the canine route. More important is the fact that we need a nasopharyngoscope with an objective at the tip of the instrument. This will permit thorough examination of both the nasal fossæ and the interior of the antrum.

2. The ostium of the maxillary sinus is usually a canal. In forty-two antra the average length of the opening into the antrum from the lateral wall of the infundibulum to the internal ostial margin measured 6.17 mm.

3. A more accurate and detailed classification of the openings into the maxillary sinus is offered, viz.: (1) Normal canal or ostium; (2) duplicate normal canals or ostia; (3) joining normal canals;

(4) joining normal and accessory canals; (5) accessory ostium or canal; (6) duplicate accessory ostia or canals; (7) joining accessory canals.

I wish to thank Dr. Baldwin and The Albany Medical College for the privilege of doing this work.

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TABLE I.

Specimen Number	Length Meatal Entry in mm.	Observations Thru Meatal Entry	Length Canine Entry in mm.	Observations Through Canine Entry	Uncinate Border to Ostial Margin in mm.	Lat. Infund. Wall to Ostial Margin in mm.	Remarks
21R.	—2.	Could not see into antrum	14.	Ostium not seen; small recess present, mallet to puncture	3.	1.	Canal is directed inf. ant. lat. Bulla is medial to infundibulum.
21L.	—4.	Could not see into sinus	14.	Ostium not seen; some mucosa viewed	13. lat.* 10. med. post.* ant.	9. 6. 2. 3. 7.	Normal ostium directed inf. post. Bulla is medial to infundibulum; does not aid in formation of infundibulum. Accessory ostium passes ant. inf. lat.
22R.	9.	Ostium recognized, mucosa well seen	28.	Ostium and mucosa well seen	10.		Canal is directed lat. and inf. Very large sinus. There is no bulla discernible.
22L.	14.	Ostium seen	20.	Ostium was seen, difficult to recognize. Mallet to puncture.	12. post.* 10. ant.	8. 6.	Canal is directed lat. and post.
23R.	2.5	Ostium was in view, not recognized	18.	Ostium was not recognized, though in field of vision	17.	9.	Ostium was a pin-head sized opening, leading inf. lat. ant. and extremely difficult to move, even after unciniate removed.
23L.	5.	Small recess appeared like ostium, which actually was not seen	7.	Very little mucosa seen; ostium not seen	14.	8.	Small ostium difficult to find, directed ant. inf. lat.
24R.	2.	Good portion of antrum seen, but ostium was not	10.	Two adjoining canal seen, mallet to puncture	17. ant.* 10. post.	9.* 2.	Adjoining canal was a recess, true ostium was directed ant. lat. inf.
24L.	7.	Normal ostium not recognized with field of vision. Accessory ostium not seen	23.	Normal ostium seen, cannot bring accessory in to view	6. ant.* 8. post.	2.* 1.	Canal courses ant. inf. lat. Accessory ostium.

TABLE I.—Continued.

Specimen Number	Length Meatal Entry in mm.	Observations Thru Meatal Entry	Length Canine Entry in mm.	Observations Through Canine Entry	Uncinate Extended to Ostium Margin in mm.	Lat. Infund. Wall to Ostium Margin in mm.	Remarks
25R.	3.	Ostium cannot be brought into view, polypi recognized post. and sup.	16.	Ostium well seen and all the polypi mentioned	10.	7.	Canal is directed ant. lat. No polyp. bulges of median wall resembled polyp. Uncinate is below inf. border of the mid. turb. Bulva cell is high above and really is not a wall of infundibulum. The 7 mm. is however measured from the floor of infundibulum.
25L.	0	A glimpse of inf. portion of antral mucosa	20.	See ostium and whole lining	10. (?)	10.	Canal is directed ant. lat. There is absolutely no bulva. Mid. turb. is poorly developed. There is no ostium above the uncinate, really post. to this process.
26R.	1.	Barely see into antrum. When post. wall of sinus removed and scope permitted to enter 2 mm., could see ostium	20.	Could see good portion of mucosa, but was difficult to see ostium which was seen from side view	14.	8	Canal directed inf.
26L.	0.5	Barely see into sinus	15.	Could not see ostium, some of mucosa seen	8. ant.* 10. post.	2.* 4.	Canal directed inf. and opened high up anteriorly.
27R.	4.	Two ostia seen, one recognized as an accessory ostium, most of mucosa seen	14.	Accessory ostium well seen, other barely. Mal-let to puncture	25. ant.* 22. post.	16. 13.	Canal directed inf. post. Second canal was ethmoid recess.
27L.	3.	Ostium a slit along with two polypi	18.	Ostium seen, not too certain	15.	9.	Canal directed inf. post. Polyp. were fragments of coagulated blood.
28R.	0.	Glimpse into sinus	23.	Ostium seen with group of polypi	20.	13.	Canal directed post. inf. lat. Three large polyp.

28L.	7.	Polypi seen, only small portion of ostium seen with extreme shifting due to obstruction by fragment of bone	23.	Ostium in field but not recognized; polypi not seen	15. post.* 9. ant.	8.* 2.	Canal directed inf. post. lat. Two polypi on floor and medial wall.
29R.	2.	Ostium seen and most of mucosa	4.	Ostium seen; mucosa also	17. med.* 14. lat.	7.* 4.	Canal directed lat. inf.
29L.	6	Gillhouse into antrum	5.	Ostium seen on extreme shifting; mallet to puncture	12.	6.	Canal directed lat. post. inf. Pair of small sinuses.
30R.	10.	Could see two ostia and most of mucosa	19.	Saw two ostia and mucosa very well. Mallet to puncture	11. post.* 9. ant.	6.* 4.	Canal directed ant. lat. Second canal was a recess leading from true ostium.
30L.	7.	Accessory ostium seen. Normal obstruction by fragment of bone	9.	Cannot see accessory ostium, normal could not be seen	13. post.* 12. ant.	7.* 6.	Canal directed lat. post. Bulla.
31R.	8.	Ostium and most of mucosa seen	16.	One ostium seen clearly, second in doubt	19.	1.	Accessory ostium directed post.
31L.	1.	Two ostia seen from side, one seen as canal	18.	Ostium and mucosa seen	12. ant.* 14. post.	12.	Normal ostium directed lat. inf. post.
32R.	1.	One ostium distinctly seen, a possible second or ethmoid recess	14.	Ostium not seen. Mallet to puncture	6.	1.	Superior accessory and inferior one directed lat.
32L.	6	Cannot see into sinus	12.	Slit like ostium seen with ethmoid ridge. Mallet to puncture	8.	6. 8.	Only one small ostium directed lat. inf. post.
33R.	4.	Ostium recognized	19.	Ostium and whole mucosa seen	5. ant.* 12. post.	1.* 8.	Only one opening into antrum. Bulla is completely medial to uncinate and really is not a part of hiatus or infundibulum. Canal directed ant. inf. lat.
33L.	9.	Ostium in view but not recognized because placed at angle	12.	Ostium is seen; mallet to puncture	17. ant.* 13. post.	8.* 1.	Canal directed inf. lat. Bulla does not form a part of infundibulum or hiatus semilunaris. Canal directed lat. ant. Canal directed lat. and post.



TABLE I.—Continued.

Specimen Number	Length Meatal Entry in mm.	Observations Thru Meatal Entry	Length Canine Entry in mm.	Observations Through Canine Entry	Uncinate Border to Ostial Margin in mm.	Lat. Inf. Wall to Ostial Margin in mm.	Remarks
34R.	0	Barely see sinus	0	Cannot see into antrum, but for glimpse laterally	post.* ant.	1.* 2.	Canal directed lat. Extremely small antrum. Bulla is medial to uncinat. There is no true infundibulum. Post. portion of uncinat. flares out into ostium of 2 by 1.5 cm., which makes up most of medial antral wall.
34L.	5.	Mucosa and ostium well seen	18.	Ostium seen and what may have been another		1. 2.	Accessory ostium. Normal ostium directed lat. Uncinate destroyed.
35R.	3.	One ostium seen, other in field but not recognized	23.	Two ostia seen, one canal like	20. post.* 23. ant.* 20.* post.	14.* 17. 20.* 16. 1.	Normal canal directed post. inf. lat. An adjoining accessory ostium directed inf. post. lat. A normal accessory ostium directed lat.
35L.	3.5	Accessory ostium could not be seen, too far forward in "scotum". Normal ostium constructed by fragment of bone	15.	One ostium seen; mallet to puncture	10.	2. 1.	Canal directed lat. ant. inf. Bulla is medial to uncinat. post.
36R.	1.	Barely see into antrum, ostium in view but not recognized. Mallet to puncture	16.	Ostium recognized; mallet to puncture	10. post.* 8. ant.	5.* 3.	Accessory ostium directed lat. post.
36L.	6.	Saw ostium and all mucosa	13.	Ostium seen from side view, not as well as through meatal puncture	5. inf.* 1. sup.	5.* 1.	Canal directed lat. There is no true bulla; hiatus really not present. Uncinate and lat. wall of mid. meatus make infundibulum, i. e., uncinat. has ostium above it which levels off with lat. wall of mid. meatus.

37R.	4.5	Ostium not recognized though in view. Mucosa a well seen	15.	Small ostium not recognized, though in field	10. post.* 8. ant.	4.* 2.	Canal directed post.
37L.	2.	Ostium cannot be seen; it is too far forward	18.	Ostium is not seen; it is in a forward recess	16.	7.	Directed lat. inf.
38R.	8.	Ostium recognized as canal	15.	Ostium is in view; mallet to puncture	19. ant.* 15. post.	11.* 7.	Canal is directed ant. inf. lat. Bulla is rudimentary.
38L.	7.	Ostium in view but not recognized	8.	Ostium in field but not recognized	16. post.* 9. ant.	9.* 2.	Canal directed ant. lat. Bulla is undeveloped and clings to mid. turb.
39R.	10.	Barely brings ostium into view, most of mucosa seen	16.	Two ostia seen	6.	2. 3.	Directed ant. inf. lat. Accessory directed inf. post. These are an adjoining, normal and accessory ostia.
39L.	7.	Ostium just seen. Two polypi seen	20.	Ostium seen; polypi also	12.	8.	Canal directed inf. post. Smooth bulge of wall appeared like polyp.
40R.	1.	Cannot see ostium	4.	Ostium is seen with small amount of mucosa	17.	8.	Ostium is a slit directed lat.
40L.	6.	Glimpse into antrum	10.	Ostium seen; mucosa also	14. med. 12. lat.	7. 5.	Canal directed inf. lat.
41R.	7.5	Two adjoining ostia recognized, a ridge separating them	12.	Adjoining ostia seen with mucosa of most of antrum	6. ant.* 8. post. 6.	1.* 2. 1.	Ant. adjoining ostium passes ant. inf. Post. adjoining ostium directed post. inf. There are two openings into antrum with one into infundibulum.
41L.	3.5	One ostium seen with ridge of probable adjacent ostium or recess	14.5	One ostium seen and mucosa of sinus clearly viewed	9. ant.* 12. post. 11.	3.* 6. 9.	Canal leads post. inf. This is post. of a pair of adjoining ostia and runs post. inf. There are two openings into antrum and one into infundibulum.

\*One wall of canal was longer than the opposite side; the mean was taken as the measure of its length.

TABLE II.

Specimen Number	Right Sided Antra				Left Sided Antra			
	Length Meatal Entry	Was Ostium Seen	Length Canine Entry	Was Ostium Seen	Length Meatal Entry	Was Ostium Seen	Length Canine Entry	Was Ostium Seen
21.	-2. mm.	No	14. mm.	No	-4. mm.	No	14. mm.	No
22.	9. mm.	Yes	28. mm.	Yes	14. mm.	Yes	20. mm.	Yes
23.	2.5 mm.	Yes	18. mm.	Yes	5. mm.	No	7. mm.	No
24.	2. mm.	No	10. mm.	Yes	7. mm.	Yes	23. mm.	Yes
25.	3. mm.	No	15. mm.	Yes	0. mm.	No	20. mm.	Yes
26.	1. mm.	No	20. mm.	Yes	0.5 mm.	No	15. mm.	No
27.	4. mm.	Yes	14. mm.	Yes	3. mm.	Yes	18. mm.	Yes
28.	0. mm.	No	23. mm.	Yes	7. mm.	No	23. mm.	Yes
29.	2. mm.	Yes	4. mm.	Yes	0. mm.	No	5. mm.	Yes
30.	10. mm.	Yes	19. mm.	Yes	7. mm.	Yes	9. mm.	No
31.	8. mm.	Yes	16. mm.	Yes	1. mm.	Yes	18. mm.	Yes
32.	1. mm.	Yes	14. mm.	No	0. mm.	No	12. mm.	Yes
33.	4. mm.	Yes	19. mm.	Yes	9. mm.	Yes	12. mm.	Yes
34.	0. mm.	No	0. mm.	No	5. mm.	Yes	18. mm.	Yes
35.	3. mm.	Yes	23. mm.	Yes	3.5 mm.	No	15. mm.	Yes
36.	1. mm.	Yes	16. mm.	Yes	6. mm.	Yes	13. mm.	Yes
37.	4.5 mm.	Yes	15. mm.	Yes	2. mm.	No	18. mm.	No
38.	8. mm.	Yes	15. mm.	Yes	7. mm.	Yes	8. mm.	Yes
39.	10. mm.	Yes	16. mm.	Yes	7. mm.	Yes	20. mm.	Yes
40.	1. mm.	No	4. mm.	Yes	0. mm.	No	10. mm.	Yes
41.	7.5 mm.	Yes	12. mm.	Yes	3.5 mm.	Yes	14.5 mm.	Yes
Total	79.5 mm.	14 Yes 7 No	316. mm.	18 Yes 3 No	83.5 mm.	11 Yes 10 No	312. mm.	16 Yes 5 No
Average Length of Entry	3.78 mm	68.7%	15.04 mm.	85.71%	3.98 mm	52.93%	14.86 mm.	76.19%
		positive		positive		positive		positive

TABLE III.

Specimen Number	Distance of Uncinate Border to Ostial Margin	Distance from Lateral Wall of Infundibulum to Ostial Margin (Length of Canal)	Direction of Canal
21R.	3. mm.	1. mm.	Inf. ant. lat.
21L.	11.5 mm.	7.5 mm.	Inf. post.
22R.	10. mm.	7. mm.	Lat. inf.
22L.	11. mm.	7. mm.	Inf. lat. ant.
23R.	17. mm.	9. mm.	Inf. lat. ant.
23L.	14. mm.	8. mm.	Ant. inf. lat.
24R.	13.5 mm.	5.5 mm.	Ant. lat. inf.
24L.	7. mm.	3. mm.	Ant. inf. inf.
25R.	10. mm.	7. mm.	Ant. lat.
25L.	20. mm.	10. mm.	Ant. lat.
26R.	20. mm.	14. mm.	Inf.
26L.	9. mm.	3. mm.	Inf.
27R.	23.5 mm.	14.5 mm.	Inf. post.
27L.	15. mm.	9. mm.	Inf. post.
28R.	20. mm.	13. mm.	Post. inf. lat.
28L.	12. mm.	5. mm.	Inf. post. lat.
29R.	15.5 mm.	5.5 mm.	Lat. inf.
29L.	12. mm.	6. mm.	Lat. post. inf.
30R.	10. mm.	5. mm.	Ant. lat.
30L.	12.5 mm.	6.5 mm.	Lat. post.
31R.	19. mm.	12. mm.	Lat. post.
31L.	13. mm.	7. mm.	Lat. inf. post.
32R.	6. mm.	1. mm.	Ant. lat. inf.
32L.	8. mm.	2. mm.	Inf. ant.
33R.	8.5 mm.	4.5 mm.	Lat. ant.
33L.	15. mm.	6. mm.	Lat. post.
34R.	1.5 mm.	1.5 mm.	Lat.
34L.	2. mm.	2. mm.	Lat.
35R.	21.5 mm.	15.5 mm.	Post. inf. lat.
35L.	10. mm.	2. mm.	Lat. ant. inf.
36R.	9. mm.	4. mm.	Ant. inf. lat.
36L.	3. mm.	3. mm.	Lat.
37R.	9. mm.	3. mm.	Post.
37L.	16. mm.	7. mm.	Lat. inf.
38R.	17. mm.	9. mm.	Ant. inf. lat.
38L.	12.5 mm.	5.5 mm.	Ant. lat.
39R.	6. mm.	2. mm.	Ant. inf. lat.
39L.	12. mm.	8. mm.	Inf. post.
40R.	17. mm.	8. mm.	Lat.
40L.	13. mm.	6. mm.	Inf. lat.
41R.	7. mm.	1.5 mm.	Ant. inf.
	6. mm.	1. mm.	Post. inf.
41L.	10.5 mm.	4.5 mm.	Post. inf.
	14. mm.	9. mm.	Post. inf.
Total	523. mm.	271.5 mm.	
Average	11.88 mm.	6.17 mm.	

## PATHOLOGY OF CHRONIC SINUSITIS IN CHILDREN.\*

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We feel that for the present the pathologic studies of sinusitis should be undertaken by the rhinologist, who has examined his patient, operated on him and removed the specimen. He must learn to study and interpret the histopathologic findings of each specimen removed. The rhinologist today reads and interprets his own X-ray plates. He must also learn to read his pathologic sections if he expects to progress in this important phase of rhinology.

The histopathology of chronic sinusitis in children does not differ materially from that of the adult, with one exception, however. In children, the involvement is less extensive, and frequently confined to one or two accessory cavities.

Clinically and pathologically we recognize two forms of chronic sinusitis, viz.: (1) Hypertrophic (hyperplastic, polypoid, etc.); (2) atrophic (fibrotic, sclerotic).

Although mixed forms of atrophy and hypertrophy exist, we feel, after considerable observation of these atrophic changes in children, that from their very incipency they are atrophic and that as such they are to be differentiated from nasal atrophy that follows chronic hypertrophic rhinitis, frequently seen in the adult.

In dealing therefore with atrophic rhinitis of the non-ozena type, we are concerned with a distinct and separate pathologic entity differing from an atrophic rhinitis associated with or following the chronic hypertrophic lesion.

Chronic hypertrophic rhinitis always begins as such, usually continuing throughout life, and in its later stages manifesting itself in the form of extensive polypoid degenerative changes of the middle turbinates, septal mucosa, ethmonasal surface (middle meatus), from which arise most of the polypi seen in this region. In the more advanced cases the pathology extends into the ethmoid labyrinth through resorption of the bony ethmoid capsule, involving the ethmoid mucosa, completely destroying the cell walls and converting

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the ethmoid labyrinth into one or two large cavities. Frequently the vault of the ethmoid is resorbed or necrosed by the constantly advancing pathologic process, giving rise to chronic pachymeningitis. It is in this type of case that a meningitis follows the removal or evulsion of an ethmoid polyp or a mass of polypi, which procedure always terminates fatally. Particularly is this common in cases where repeated ethmoidectomies have been performed.

When examining a patient rhinoscopically, it is not at all difficult to tell from gross appearances of the nasal tissues what the predominating lesion is. Either it tends toward hypertrophy or toward atrophy. The nasal mucosa and its turbinates, being a mirror picture of the sinus mucosa, reflects the pathologic state of the latter. Both types of sinusitis may be associated with a superimposed suppuration. Vasomotor and allergic rhinitis will not be included in the present discussion. We have never seen an allergic rhinitis with an atrophy of the nasal mucous membrane.

1. *Hypertrophic Sinusitis*.—(a) Gross pathology: The gross pathologic picture is that of a general hypertrophy of the nasal mucosa accompanied by congestion and turgescence and loss of tone. The inferior turbinate bodies are mostly involved and are found greatly enlarged, impinging on the nasal septum, resulting in complete nasal obstruction of one or both nares. When superimposed by suppuration, the nasal chambers are filled with mucopus or pus bathing the turbinates. On shrinking the nasal mucosa, with adrenalin solution (1-1000), the middle turbinates come into view. These are found congested and hypertrophic but to lesser degree than the inferior turbinates. In the presence of an ethmoid infection the anterior tips of the middle turbinates are covered with pus or a dried, glistening film of secretion resembling a crust, on the removal of which the mucosa frequently present polypoid changes. Secretions can frequently be seen in these cases, coming from the region between the ethmoid and middle turbinates—i. e., upper middle meatus from hiatus semilunaris, nasofrontal duct or hiatus antra. The pus filling the nose can be seen dripping posteriorly into the epipharynx and into the throat and larynx. Examination of the throat reveals a marked postnasal drip of pus, a markedly irritated, congested pharynx diffusely infiltrated with lymphoid nodules, particularly in the fossæ tonsillaris, where a previous tonsillectomy has been done, as well as large pieces of recurrent tonsil tissue. Laryngoscopy, when possible, frequently reveals dripping of pus into the larynx through the interarytenoid space or, filling at first the pyriform sinus, drips into the larynx by spilling over the arytenoids.

(b) Microscopic pathology: Due to the length of this paper, brief reference only will be made to the microscopic pathology. The mucosa lining the nasal chambers and the accessory sinuses as well as its underlying bony framework has a definite architectural structure. This structure shows characteristic variations in accordance with its site or location. The pathologic changes, however, are much the same in the majority of cases, differing somewhat in instances, according to the degree of disturbed circulation, drainage and ventilation. For the sake of brevity, therefore, and due to this general similarity in the pathology, the latter will be discussed more from a general aspect and in accordance with the changes found in the various important layers comprising the nasosinal mucosa.

1. Epithelial layer.
2. Subepithelial, hyalin layer or basement membrane.
3. Tunica propria (stroma).
  - (a) Blood vessels.
  - (b) Glandular structure.
  - (c) Cellular elements.
4. Periosteum.
5. Bone.

The subdivision of chronic hypertrophic sinusitis and rhinitis into papillary, edematous, infiltrative, cystic, degenerative, polypoid, granular, etc., is, to my mind, superfluous and unnecessary. One rarely sees a single specimen without being able to find almost any or all of the above forms; and if sufficient sections are made at various levels of several pieces of tissue removed from different parts of the nose and sinuses in the same individual, one is sure to find every type of lesion possible, of course with certain exceptions, from the absolute normal to the most pathologic. It is also true that certain pathologic lesions will predominate, such as extensive polypoid degeneration of mucosa with marked cystic changes in them, marked edema of stroma, papillary hypertrophies, areas of intense round cell infiltration, extensive fibrosis and degenerative changes in the gland and epithelium structure, etc., but all these grades or forms are but the result of the same pathology, only in a progressed and altered state, and still fall under the head of chronic hypertrophy. According to Manasse, a sharp differentiation of these forms is not possible, because there are many mixed forms, indeed cases where in one and the same specimen all forms are united.

1. *The Epithelial Layer* (a).—The epithelium of the nasal and turbinal mucosa shows moderate to marked hyperplasia. Where poly-

poid changes exist the epithelial layer may be hypertrophied to the extent of ten to twelve layers of columnar epithelium piled up on one another with many infiltrating leucocytes making their way to the surface. Goblet cell formations are rather common at the surface, where they become clumped with surface secretion and bacteria. Areas with exfoliation of epithelium may be found but are infrequent. Due to exposure and irritation to air and secretions, metaplasia of the epithelium is rather common, particularly when advanced polypoid changes have occurred and where drainage and ventilation are poor, as in the olfactory fissure and outer surface of the inferior and middle turbinates. It is very common to find that hyperplasia is frequently confused with metaplasia and so reported erroneously, due, perhaps, to the fact that the rhinologist or pathologist is not thoroughly acquainted with the difference between the two. Ulceration of the epithelial layer is very uncommon and even rare, but does occur, more often in the hypertrophic suppurative type than that of the atrophic type.

(b) Sinus epithelium differs essentially from the nasal type in that it is a simple columnar ciliated type, consisting (1) in the ethmoid mucosa of columnar ciliated cells intermingled with olfactory and sustentacular or supporting cells. The olfactory cells lie between the sustentacular cells. Their nuclei are spherical, lie at different levels and more deeply placed than the sustentacular cells, and form the zone of round cell nuclei, and (2) in the antra, sphenoid and frontal sinus, the mucosa is of simple columnar ciliated epithelial cells, many of which are of the secretory or goblet type. The goblet type predominates, particularly where hypertrophic and polypoid degenerative changes abound. Here we find them in very great numbers, replacing the columnar surface epithelium, where they seem to take on great secretory activity, due very likely to the presence of very few serous or mucus glands in the stroma or subepithelial layer. Hyperplasia of the epithelium in the ethmoid was not noted. In the antrum hyperplasia was rather common. Loss of cilia, or exfoliation or desquamation of epithelium in the sinuses is uncommon. Metaplasia is frequently found. Ulceration of the surface of the epithelium is rare and has not been noted in these studies of sinus epithelium, except in one instance, although some authors do report their presence.

2. *The subepithelial layer, hyalin layer or basement membrane*, found in the mucous membrane of the nose and turbinates and rather faintly present if at all in the sinuses is worthy of note. It is thought to be derived from the underlying connective tissue. It



is composed of a dense but homogeneous layer of collagenous fibers, very faintly or totally unstainable, on which the epithelial cells appear to rest, and through which spaces or canaliculi migrating leucocytes are found working their way to the surface. It is always found in the mucosa of the nose as a rather distinct and sharp layer and forms one of the differentiating points between nasal and sinus mucosa. It is occasionally found as a distinct layer in antral mucosa but rare in the ethmoid, sphenoid or frontal. When present it is found associated with chronic inflammatory lesions both of the hypertrophic and atrophic forms, as well as in allergic disturbances as asthma and allergic rhinosinusitis. In chronic inflammations, particularly of the atrophic form, it is found greatly thickened with epithelial exfoliation, and seems to act as an important barrier or protecting agent against entrance of infection to the deeper layers of the mucosa. Some do not, however, consider it to have any clinical or pathologic significance other than being an evidence of chronic epithelial irritation.

3. *The tunica propria* or stroma consists of loose connective tissue and blood vessels with infiltrating cellular elements, the latter varying in accordance with the degree and character of inflammatory changes present. In the nasal mucosa, particularly the turbinates, we must consider the glandular and cavernous layer. In the former is found many serous and mucous glands, and the latter consists of cavernous blood channels surrounded by a network of elastic tissue which enables the sinuses to fill and empty rapidly. The middle turbinate differs from the inferior in having fewer glands with predominating mucous glands, while in the inferior turbinate serous glands predominate. The middle turbinates have no cavernous blood spaces. Cystic degeneration of mucous and serous glands is commonly found particularly where polypoid changes are well advanced. The presence of edema causing a blocking of secretion in the ducts and the latter to become distended with mucoid material. The cysts assume frequently large proportions and become filled with cellular elements through invasion of a great number of leucocytes. On reaching the surface, they rupture and empty their contents on the surface epithelium. The glands are lined by a single layer of cuboidal epithelium. When blocked up, the cells secrete more actively and assume goblet cell structure.

The blood vessels are thickened and increase in number in accordance with degree of hyperplasia or hypertrophy. Proliferative changes of the layers are common, particularly the endothelium which, causing obstruction to the blood flow, increase stagnation and

edema of the surrounding connective tissue. Thrombosis of blood vessels in this form of sinusitis is rarely if ever seen.

*Cellular Elements.*—Round cell or lymphocytic infiltration with polymorphonuclear cells, is most common. Many eosinophiles and plasma cells are found, particularly in the allergic types of hypertrophy. The plasma cell is frequently mistaken for the eosinophile, and should therefore not be confused with one another.

Round cell infiltration varies in accordance with the amount of infection and local irritation. Areas of dense infiltration, both subepithelially and around gland structure, is very common, particularly in the chronic polypoid type where suppuration is extensive. Areas where mucosa is densely infiltrated with cells to the point of pus formation is common, particularly in the advanced forms of antrum and ethmoid suppuration. Here cysts filled with pus and subepithelial collection of purulent secretion are common. Finck refers to the former as mesothelial cysts.

The antral tunica propria differs from that of the nose in the absence usually of a hyalin layer and presence of only a few secretory glands. Polypoid degeneration of the mucosa is very common with the predominant presence of cystic degeneration of glands. Marked round cells and leucocytic infiltration are also outstanding features.

The ethmoid tunica propria has very few secretory glands. Here the secretory function is carried on by the epithelium, which assumes goblet cell activity, secreting large quantities of mucoid secretion admixed with many lymphocytes and leucocytes that find their way to the surface through the basement layer.

The outstanding lesion of the tunica propria itself is usually a marked edema with resulting low grade minute polypoid or at times cystic degeneration with rarely polypi of any large size. The latter are rather rare within the ethmoid labyrinth. Polypi coming from the ethmoid are usually polypoid degenerations of the nasal mucosa lining the ethmoid or outer wall of the middle or superior meatus and rarely coming from the ethmoid mucosa.

However, with the presence of necrosis or resorption of the medial ethmoid wall small polypi from the ethmoid may extrude into the nose and assume large proportions, but this is rather rare. When the latter does occur they constitute the dangerous type of case, for in removing these polypi surgically one never knows where they come from. As it is possible that they may have their origin from the ethmoid vault and in avulsing these polypi injury to the dura is possible. This type of case can be termed the dangerous type

and when operated on frequently develop meningitis and terminate fatally. More will be said about this form of sinusitis when discussion of the bone pathology will be taken up.

4. *The Periosteum.*—The periosteum is derived from the connective tissue of stroma and is at times scarcely demonstrable. When present it may vary from a very finely defined bone covering to a markedly thickened and densely infiltrated structure, depending usually upon the degree of pathology present in the overlying tunica propria. The same cellular elements will be found infiltrating both the periosteum and tunica propria. Subperiosteal hemorrhage, both old and recent, are commonly seen and are frequently of traumatic origin. Edema causing wide separation of the periosteal fibers is frequently seen. Normally, the periosteum in the ethmoid, sphenoid and frontal sinus is scarcely discernible, so that unless the lesion is of long standing, one cannot definitely demarcate between the tunica propria and a periosteal layer, the former here taking the place and function of a periosteal layer.

5. *Bone.*—The bone pathology constitutes the most interesting and most difficult phase in the pathology of chronic sinusitis. The pathologic bone changes met with are never of one or another distinct variety, but rather a mixture of various multiple pathologic processes going on simultaneously and one alongside another.

The most frequent and common lesions noted were: (a) Bone rarefaction, resorption or atrophy; (b) bone fibrosis; (c) bone necrosis; (d) bone hypertrophy.

(a) Bone rarefaction, resorption or atrophy is most commonly seen. The process may be seen to begin as bone erosion, frequently due to chronic periostitis of the ethmoid septa or the naso-antral wall, both middle and inferior meatuses. This process is evidently due to the presence of bacteria in or subperiosteally. This process of atrophy by erosion is rather slow and drawn out.

Bone atrophy is also very commonly seen associated with fibrosis and proliferative fibrous connective tissue changes. Connective tissue proliferation may be (1) endogenous—that is, begin from within the bone spaces through proliferation of connective tissue about the blood vessels, and (2) exogenous, or connective tissue proliferation outside of the bone, i. e., periosteum or stroma. Here primarily it starts with an erosion of the bone and subsequent invasion of bone by connective tissue. Thus thinning out of the bone takes place both from within and without, causing rapid bone resorption or atrophy. Fibrous tissue strands may be seen splitting the bone into

long separate longitudinal strands. Bridges of connective tissue may be seen separating islands of bone from one another. In case of the ethmoid septa, they ultimately become entirely resorbed, converting the ethmoid labyrinth into one or two large cavities. In case of the naso-antral wall, the latter is frequently converted into a thin fibro-membranous partition. Atrophic and fibrotic changes are commonly seen associated with extensive hypertrophies and polypoid degenerative changes in nasal structures, prominently in the middle and occasionally in the inferior turbinates. Here the process is largely a mechanical one, of course, associated with bacterial infection, etc. Endarteritis and vascular thrombosis, causing a shutting off of blood supply, is the chief cause of atrophy. The bone resorption here is very slow. The bone cells lose their sharp outline, appear pale and swollen, nuclei enlarged and pale, staining rather poorly at first and later not at all. The cells later fade and disappear, leaving empty spaces in the bone. The bone itself takes on a poor stain, looks pale and faded, gradually losing its outline until completely resorbed and its place taken by young connective tissue. This lesion is best studied in polypoid degenerative changes of the anterior half of the middle turbinate bone.

(b) Bone fibrosis, as outlined above, is found in all advanced cases of chronic hypertrophic lesion associated particularly with suppuration. It is found associated with bone resorption, softening and atrophy. Also where pressure atrophy takes place due to polypoid hypertrophies, etc., fibrosis is present in nasal pathologic lesions associated with infections, asthma and the so-called allergies. Fibrotic changes are frequently met with in chronic ethmoiditis where complete resorption of the ethmoid capsule takes place, involving the ethmoid vault, and finally the dura of the anterior cranial fossæ. The latter type constitutes the dangerous and frequently fatal cases when operated upon.

(c) Bone necrosis is not as commonly seen as the above lesion just described and may even be considered as an infrequent finding. Necrosis was most frequently found in chronic antrum suppuration of long standing and involved chiefly the naso-antral wall. The lesion is considered as being caused by necrosis of the wall due to the involvement of perforating blood vessels, arteries or veins by an infected thrombus, the infection spreading through the mucosa lining the antrum, its wall and involving the nasal side. Although to a lesser extent necrosis is next in frequency seen involving the paper plate with exposure of the orbit and its contents to infection. When not disturbed by operative procedure, the latter rarely takes place,

although rupture of abscess pockets of the ethmoid into the orbital cavity is occasionally seen. The floor of the frontal and anterior sphenoid wall are other sites where necrosis occurs. The necrotic bone areas are eventually absorbed and replaced by fibrous connective tissue or new bone formation. In the latter the osteoclasts, multinucleated cells, or the so-called giant cells, play an important rôle. They are found in the areas where bone repair goes on or in bony dugouts known as Howship's lacunæ. It appears that due to irritation set up by infection, the osteoblast is capable of assuming the function both of the builder (osteoblast) and the destroyer of bone tissue (osteoclast). The osteoblast is said to be derived from either the periosteum or fibrous connective tissue of the tunica. The fibroblasts or young connective tissue cells lining the marrow cavities may also take on the function of both osteoblast and osteoclast. Von Gazer considers this metamorphosis of the osteoblast into the osteoclast as only functional and caused by the release of paraplasmic substances from dying bone. Under normal conditions the osteoblasts appear as a line of dark stained spindle shaped singly nucleated cells, lined up end to end in regular order and parallel to the bone surface underneath the periosteum. Under deranged or pathologic states they lose their clear and well defined outline, appearance and arrangement, standing up on edge, become irregularly enlarged, assume different forms and set to work as osteoclasts or bone destroyers causing gradual erosion of bone surface by eating away the latter and ultimately absorbing the bone completely. Fibrous connective tissue ultimately displaces the bone, as previously described under bone atrophy and resorption.

(d) Bone hypertrophy is rather uncommon as a widely distributed lesion. It is, however, occasionally found in chronic hypertrophic lesions involving chiefly the inferior turbinate. The bony turbinal lamina of the inferior turbinate is normally thin and cribriform with the periosteum dipping down in these depressions and tightly adherent to them. Under chronic irritation and periostitis associated with chronic nasal suppuration and infection, the inferior turbinal bone (lamina) is found to be increased many times its normal thickness, frequently becoming so obstructive to respiration that nothing but a complete resection of this bone submucously will relieve the obstruction. Associated thickening of the adjoining sinus walls are seen. This bony overgrowth is often of hard sclerotic character, making it exceedingly difficult to penetrate or resect the nasal wall of the antrum, sphenoid or frontal floor.

2. *Atrophic Sinusitis*.—(a) Gross pathology: The gross pathology shows a decided roominess in the nose, involving one or both

nares, most frequently both. The roominess may vary from a moderate to a marked degree, where both nasal chambers appear exceedingly wide, exposing to direct inspection the epipharyngeal wall and soft palate. In the advanced cases marked atrophy of the inferior and middle turbinates are present. When suppuration is present, pus, mucopus or crusts may be seen filling the nasal fossæ and extending or dripping posteriorly into the nasopharynx. The mucosa of the middle turbinate is often covered with a thin, glassy looking secretion. During operation the middle turbinate is frequently found adherent to the naso-ethmoid mucosa (middle meatus), with low grade polypoid degenerative changes of mucosa between the two. Free pus is frequently found in this region, particularly on opening the ethmoid cells. The pharynx is usually atrophic, dry and glazed, due to film of purulent secretions covering it. Crusts and thick pus may be found, extending down into the larynx. Extensive polypoid changes are never seen. The polypi often seen protruding from underneath the middle turbinate and ethmoidal region are impoverished-looking specimens and never attain any large proportions.

(b) Microscopic Pathology.—1. Epithelial layer: The epithelial layer of the nasal mucosa and turbinates shows usually a marked dissolution, exfoliation or complete absence of surface epithelium. In earlier cases the epithelial lining, although present, exhibits an unhealthy unevenness interspersed by areas of complete denudation, hyperplasia, metaplasia or clumping together of epithelium, cellular elements, bacteria and mucoid secretions. In advanced cases a complete loss of epithelium is most common. In the sinus epithelium degeneration and exfoliation are less marked than in the nasal chambers. Metaplasia with complete changes of columnar type of cell to the cuboidal or flat stratified and horny epithelial cell is quite common, in the nasal mucosa where it is exposed, due to roominess, to swift currents of air and irritating substances, such as dust or fumes, carried in by it. True ulceration of the surface epithelium, although occasionally met with, is considered rare.

2. Subepithelial or hyalin layer is usually prominently present and thickened, particularly when exfoliation of the surface epithelium is marked or entirely absent. Cellular infiltration, particularly of the round cell variety, is commonly seen diffusely infiltrating this layer and coming from the stroma below it.

3. Tunica Propria.—This layer, as in the hypertrophic type, consists of fibrous connective framework, glands, blood vessels and cellular elements infiltrating it. The general appearance of the



stroma is that of a low grade chronic inflammatory process, varying according to the length of time the condition has been present, and whether or not it has been superimposed by infection as well as to how recent the last attack of suppuration took place. Opinions vary among authors and pathologists regarding the relationship of simple atrophy to fibrosis and fibrous degenerative changes associated with ozena.

The common lesions found are round cell infiltration, in and around glandular tissue; glandular atrophy, due largely to compression of surrounding fibrosis in the more advanced cases; localized areas of intense round cell infiltration; new blood vessels and fibrosis. Blood vessels are frequently seen to be markedly thickening, particularly in their outer coat. Perivascular lymphocytic (round cell) infiltration is commonly seen. Thrombosed blood vessels are rarely if ever seen.

4. The Periosteum.—The periosteum is often thickened and infiltrated with the same cellular components involving the stroma next to it. In chronic and well advanced cases, bacterial stains show clumps of bacteria involving the periosteum or between the periosteum and bone.

5. The Bone.—In characteristic well defined atrophic cases of long standing the skeletal structure of the nose and paranasal sinuses is that of a well defined subinvolution form characterized by retarded or underdevelopment of the nasal sinuses, associated with a diffuse sclerosis of its bony framework. The X-ray examination in these cases shows a decided underdeveloped, infantile type of sinuses in the older children as well as in the adults. The sinuses are small, poorly illuminated, with the frontal frequently absent. The ethmoids are sclerotic in type and very poorly developed and aerated when present. The outer nasal wall is markedly thickened and turbinal bony framework atrophic. The skeletal lesion in these cases is very much similar to the atrophy with ozena. Although occasionally one encounters advanced simple atrophy in children with large, well developed sinuses, the reverse, however, is the rule. Microscopically, bone atrophy, sclerosis and bone hypertrophy are the predominating lesions.

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LXVII.

THE ETIOLOGIC FACTORS IN THE FORMATION OF  
CHOLESTEATOMA.\*

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The presence of cholesteatoma complicating a chronic suppurative otitis media has long been considered a cardinal indication for radical surgery. However, in many instances the patients will refuse to undergo a major operation for what they consider to be a minor ailment, in spite of protestations by the doctor of the potential seriousness of the infection. It has, therefore, been found necessary to treat these patients conservatively, whether or not surgery would seem to be indicated. The good results frequently obtained by treating these patients have led us to advocate conservative treatment for many of the cases with cholesteatoma on whom we formerly advised the employment of radical surgery.

The principles upon which our treatment is based are predicated on our theory of the cause of the development and growth of cholesteatoma. (In our consideration of cholesteatoma we do not include the true congenital tumors, but only the pseudocholesteatomata which occur in the ear.)

Whether the cholesteatoma is primary, arising from an invagination, pouching and rupturing of Shrapnell's membrane following the formation of adhesions walling off the attic from the middle ear in an infantile otitis of the catarrhal type, or is secondary to a suppurative otitis with an ingrowth of the epidermis of the external canal through a marginal perforation into the middle ear and attic, its development is the same, though the suppuration is a result of the former and the cause of the latter.

It is generally accepted that cholesteatoma is formed from the desquamated epithelium of the epidermis which has grown into the tympanic cavity from the external auditory canal, the most reasonable explanation of which is that of Wittmaack,<sup>1</sup> who considers the process as one of attempted repair by nature, the squamous epithe-

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\*Presented before the annual meeting of the American Laryngological, Rhinological and Otological Society, Charleston, S. C., April 3, 1934.



lism of the external canal growing into the middle ear to replace the degenerating mucous membrane. The formation of cholesteatoma will only occur if there is an inadequate outlet for the drainage of the exfoliated material. The attic and antrum are ideal localities for this formation, as they have a relatively small outlet to the middle ear. In cases where there is a blocking off of these spaces from the middle ear complete healing of the middle ear may occur without invasion of the epidermis into the attic and antrum. Conversely, when there is a relatively large opening into the attic and antrum, epidermatization may occur and healing result without cholesteatoma formation, but only if there is an adequate outlet for the discharge and elimination of exfoliated material.

The malignant effect of moisture upon cholesteatoma has long been recognized by the profession and several reasons ascribed for it, the most acceptable one being that cholesteatoma has marked hygroscopic powers and, therefore, the presence of moisture will cause the mass to swell and exert increased pressure on the surrounding walls; also that the presence of moisture affords a more favorable medium for bacterial growth. While these effects are probably true, it would seem that the most malignant effect of all has been entirely overlooked, namely, the effect of moisture on the actual growth of cholesteatoma.

It is our belief that the primary and major factor in the development and growth of cholesteatoma is the presence of moisture. Whether the moisture is due to suppuration, is a transudate, an exudate or even water, the effect is the same. The surface epithelium of the body does not tolerate moisture well, and the continued contact of moisture causes irritation and desquamation of epithelium. The squamous, stratified epithelium, which grows into the tympanic cavity and gives rise to cholesteatoma, is surface epithelium. The desquamation of epithelium and formation of cholesteatoma is primarily due to the constant presence of moisture bathing the epithelium.

Even in the primary cholesteatoma, with ingrowth of the epidermis through a perforation of Shrapnell's membrane, the same conditions are present. At first there is a transudate in the attic due to the negative pressure which existed before the rupture of the pouch. As epidermis grows into the attic and comes in contact with the hyperplastic mucosa, there occurs an exudate from the mucosa due to the irritation of two foreign types of epithelium brought into juxtaposition. Until the epidermis entirely replaces the mucosa this irritation continues and with it the moisture. To this may be added the suppuration which usually occurs from secondary infection. Dur-

ing the whole process of epidermatization of the cavity the epidermis is constantly irrigated and undergoes continuous desquamation. Moreover, there is only a small opening communicating with the outside, evaporation is limited, and the cholesteatoma itself has the property of absorbing and retaining moisture. Of course, the presence of moisture is not the sole cause of desquamation, but we believe it to be the primary one. Inflammatory reaction to infection, chemical irritation from the fluids, the biologic reaction of two foreign types of epithelium coming into juxtaposition and mechanical pressure from the mass of cholesteatoma are all added factors in causing desquamation.

The cases of dry cholesteatoma, which are occasionally seen, can be explained on this basis and may be either primary or secondary. The primary type, in which the attic and antrum are walled off from the middle ear proper, we believe, are cases of moist cholesteatoma during their development. However, after the mucous membrane of the attic and antrum is entirely replaced by epidermis and no longer causes any secretions, the moisture present may gradually evaporate and, in the absence of secondary infection, a completely dry state may result. The same condition may occur with a secondary cholesteatoma, but only if the entire cavity becomes epidermatized and there is no secretion from the eustachian tube. It is very significant and in support of our theory that these cases may remain dormant for years, with no discharge and no increase of the cholesteatoma, until the introduction of moisture stirs them to renewed activity. Ruttin<sup>2</sup> reports several such cases in which an acute otitis was superimposed on a dry cholesteatoma. Water in the ear from swimming or bathing is often the causative factor in changing a dry cholesteatoma to a wet and active one, or in causing the new formation of cholesteatoma in an ear which may have been dry and clean for a period of years.

Might this not also be an explanation of the fact that some cases of chronic suppuration with cholesteatoma may go for many years, or even a lifetime, without developing any complications, while in others a large cholesteatomatous cavity with perforation may occur in a relatively short time? The history of these cases is in itself suggestive. The story is often not one of a continual discharge but an intermittent one with an occasional report that the ear had not discharged for some years at a time. Could these not be cases of a dry and dormant cholesteatoma, which intermittently became active due to the introduction of moisture? It was formerly our belief that these cases had a constant, slight discharge with evaporation before

it reached the external meatus and that the patients were simply unaware of it.

It is significant and in support of Wittmaack's<sup>3</sup> theories of pneumatization, that, in most cases of cholesteatoma, primary or secondary, there has been a lack of pneumatization of the mastoid, and the altered mucous membrane in the middle ear presents a favorable soil for the development of ingrowing epidermis. In other words, the occurrence of cholesteatoma is largely dependent on the altered mucosa of the middle ear, and a sclerotic mastoid is the result of maldevelopment and not of the cholesteatoma.

Almour<sup>4</sup> brought out this point and further showed that in operating on these cases it was unnecessary to remove this sclerotic bone but simply to clean out the middle ear, antrum and diseased areas. Healing was more rapid in cases in which the cholesteatomatous matrix was not removed, since it contains the elements for the development of epithelium over the denuded areas.

Moreover, Kopetzky<sup>5</sup> has pointed out that the healed radical cavity with its lining of epidermis is the same as the cholesteatomatous lined cavity which nature has been trying to heal, or, in other words, that what the surgeon accomplishes with the use of a skin transplant, nature precedes him in doing by causing an ingrowth of squamous epithelium to cover or replace the diseased areas. It is preferable for the otologist to try and assist nature in accomplishing this end than to resort to radical surgery to replace nature's efforts. We believe surgery should be resorted to only if our efforts to assist the natural healing process have failed, or if there is evidence of labyrinthine or intracranial extension.

How can we best assist nature's attempts at epidermatization? By helping to remove those factors which inhibit the growth of epidermis and by helping to remove the waste products. Moisture is irritating to skin but not to mucous membranes. The reverse is true of dryness. Therefore, by the use of anhydrous or dehydrating solutions we help the process of epidermatization at the expense of the mucosa, and the shrinking effect on granulations makes nature's efforts to cover or replace them with epidermis so much the easier. The desquamation of epithelium and consequent formation of cholesteatoma is also diminished. Secondly, we can help the elimination of waste products, especially of cholesteatoma, by instrumentation and lavage, and by maintaining a free opening for the escape and drainage of these waste products.

The use of anhydrous or dehydrating solutions has long been advocated in the treatment of chronic suppuration with chole-

teatoma, but the effect of the treatment is frequently spoiled by the preliminary use of peroxide or other aqueous solutions. The solution usually employed is alcohol, but unless it is at least a 95 per cent solution its dehydrating effect is lost. Rejto<sup>6</sup> recently recommended the use of completely anhydrous solutions and reports good results from the use of carbon tetrachloride.

The series of cases which we are reporting here is not a series of selected cases but a series of fifty consecutive cases seen by us in private practice during the past nine years. The only cases not included are those concerning whom our records are incomplete and from whom we have been unable to obtain follow-up data.

Conservative treatment was advocated only in those cases which showed no signs or symptoms suggestive of involvement of the labyrinth or meninges. Particular attention was paid to the presence of dull, unilateral headaches, changes in disposition and mental fatigue or forgetfulness, as we have found that these symptoms often accompany an exposure of the dura, with or without an extradural abscess. We believe the radical operation is clearly indicated in any cases of chronic suppuration with cholesteatoma which have dull unilateral headaches which do not promptly disappear following a short period of conservative treatment.

A radical mastoidectomy was performed on fourteen cases in this series, all of whom have clean, dry cavities. There was an open eustachian tube in two of these with occasional mucoid secretion when they caught cold, but this secretion was easily controllable with alcohol. Of the fourteen cases operated on, eleven showed symptoms or signs of extension and in ten of these this was confirmed at operation. Of the ten cases with extension of infection, nine had dull unilateral headaches, varying from the frontal region to the occiput.

Conservative treatment in this series was based on the following objectives:

1. To establish and maintain a free drainage outlet;
2. To eliminate the cholesteatoma present;
3. To eliminate granulations and promote epidermatization;
4. To dry up any secretion present.

The method of treatment was briefly as follows: Any polyps or granulations were removed by snares, punch forceps, curettage or caustics. If granulations sprang from a carious ossicle it was removed. Otherwise, unless the ossicular remains showed active necrosis or definitely impeded free drainage, they were left alone. It was

rarely found necessary to enlarge even a small Shrapnell's perforation. Attic and antrum lavage was carried out with a 5 cc. syringe and attic cannula, using only 95 per cent alcohol or ether, and was followed by suction with a small pipette or cannula. By this means large masses of cholesteatoma could be washed loose and sucked out. This procedure is somewhat disagreeable to the patient because of the vertigo aroused by the caloric effect of the alcohol or ether, and also by the negative pressure set up by suction. The reaction from the former can be reduced by first warming the alcohol. This procedure could usually be repeated two or three times at one sitting without ill effects on the patient, and has been well tolerated even by young children, though a primary anesthesia with ethyl chloride was sometimes used with them. To be successfully carried out, this procedure requires very delicate manipulation. The cannula should be flexible so as not to cause any trauma if the patient jerks. It should be of fine caliber so that there may be considerable force to the stream of alcohol or ether forced through it, and its curved tip should be as short as possible so as to facilitate its passage through a perforation and its rotation in any direction desired. In only one case on whom we used this treatment have we ever had an unfavorable reaction. That patient, following the first treatment with an attic lavage, had vertigo and nystagmus for several days. A subsequent radical mastoid operation showed no evidence of a fistula, though there was a positive fistula test after the treatment which was not present before. In this series of cases office treatments were carried out weekly or biweekly when possible, and, during the intervals, the patients used 95 per cent alcohol as ear drops twice a day, massaging the tragus after instillation so as to force the solution well into the middle ear. The diagnosis of cholesteatoma should not be difficult if the anhydrous treatment and attic lavage are employed on all cases of chronic supuration with marginal perforations. If present, macroscopic masses of cholesteatoma can nearly always be seen in the washings on the second visit if not the first. Moreover, any case in whom epidermis has grown into the tympanic cavity should be treated the same way, as a cure will result only if the epidermis can completely replace the mucosa lining the cavity.

Of the thirty-seven cases in this series treated conservatively thirty-one have dry ears which we consider cured, though no case was considered cured until the ear had remained dry for six months and repeated attic lavage resulted in a return of only a clear solution. The average number of treatments required to obtain dry ears in this group was between nine and ten over a period of five to six months.

Six cases were failures in spite of prolonged and thorough treatment. Three of these six failures were in children with open eustachian tubes; in fact, in only one case out of four were we able to get a dry ear following treatment in children. Of the other three failures, two have had dry ears for from four to six months at a time, but recurring tubal discharge with every head cold has each time resulted in a return of cholesteatoma. The last case is a puzzler. When first seen she complained of deafness and tinnitus and claimed her ear had not discharged for years. Examination revealed a large, dry anterior central perforation and also an anterior perforation of Shrapnell's membrane plugged with a large dry cholesteatoma. The attic appeared completely walled off from the tympanic cavity. The cholesteatoma disappeared after a few treatments. A few months later this patient returned with a clear tubal discharge coming from the eustachian tube, but with the attic perforation entirely closed. This status has continued for two years, but she has been entirely free of symptoms, except for occasional moisture in the ear coming from the eustachian tube. The fact that the failures from treatment in this series all had wet, secreting eustachian tubes is a very significant one and supports our theory as to the role played by moisture in the development of cholesteatoma.

Recurrences are common in healed cases and may be expected to occur at any time if moisture gets in from the outside or by way of the eustachian tube. The latter is especially liable to happen in conjunction with head colds if the eustachian tube is open. In the present series of cases there were recurrences in thirteen of the thirty-one dry ears. However, if attended to, they rarely required more than one or two office treatments and alcohol drops for a couple of weeks before returning to a dry state.

It is of great importance that these cases report for periodic examinations at least two or three times a year. They must be cared for like a postoperative radical cavity, but because of the smaller cavity present and small opening to the exterior they must be watched more closely. The perforations frequently become blocked by dried scales or pieces of epidermis which should be removed whenever found. For the past few years I have advised these cases to put a few drops of alcohol in the ear every week or two as a prophylactic measure.

It may be argued that these dry ears are not really healed but are harboring infection and that cholesteatoma is still present. In the first place, repeated attic lavage with alcohol fails to wash out any cholesteatoma and the solution remains clear. Secondly, the findings

on two of the cases in this series are significant. The first case was a woman of 27, giving a history of discharge for years, cause unknown. Examination showed an epidermatized middle ear but the attic was occluded with cholesteatoma. The ear became dry after treatment for six weeks and remained dry for the next year. This woman developed an anxiety neurosis and wanted her ear operated on because she was afraid of developing a brain abscess. A radical mastoid was performed by another surgeon with negative findings. The antrum and attic were clean and dry. The second case was a young man with a chronic attic suppuration and much cholesteatoma, who was treated by us. His ear had been dry and without any recurrence for four years when he was injured in an auto smash-up. He had a fracture of the base of the skull with blood and spinal fluid escaping from the ear; also vertigo and nystagmus of one month duration. He was not seen by us until four months later. Examination at that time showed a large mass of cholesteatoma again occluding the attic and a positive fistula test. The ear became dry again after one month of treatment and has remained dry for a year and a half since. He has fair hearing and no longer gives a positive response to the fistula test. His ear must have been dry and clean at the time of the injury or he probably would have developed a meningitis. On the other hand, the escape of blood and spinal fluid into the ear started a renewed desquamation of epithelium, with the result that he had a well developed cholesteatoma four months later.

#### SUMMARY AND CONCLUSIONS.

Moisture is the primary and major factor in the development of cholesteatoma.

Only anhydrous solutions should be employed in the presence of cholesteatoma.

Conservative treatment should be tried only in the absence of symptoms or signs of extension beyond the confines of the middle ear and mastoid.

Cures can be obtained only if there is complete epidermatization of the walls of the cavities.

The presence of moisture will prevent cures and cause recurrences. This is especially true if granulations or an open eustachian tube are present.

Conservative treatment is rarely successful in young children because of tubal secretion.

Periodic examinations are important after the ear has become dry.



The early recognition and proper treatment of chronic otitic suppuration with cholesteatoma should obviate the need of radical surgery and prevent the serious complications which so commonly occur at present.

121 UNIVERSITY PLACE.

#### CHART I.

Total number of cases .....	50
A. Number of radical mastoids .....	14
(1) Extension of infection to dura or labyrinth .....	10
(2) Healed radical cavities .....	14
(a) Open eustachian tubes .....	2
B. Number of cases treated conservatively .....	37
(1) Dry following conservative treatment, 31, or 84 per cent.	
(a) Average number of treatments, 9 to 10.	
(b) Average duration of treatment, 5 to 6 months.	
(c) Average duration of dryness, 3 years.	
(2) Failures following conservative treatment .....	6
<i>Reasons for failures:</i>	
1. Child. Tubal discharge.	
2. Child. Tubal discharge.	
*3. Child. Tubal discharge, granulations in aditus.	
4. Adult. Tubal discharge.	
5. Adult. Tubal discharge, probably secondary, probable granulations in antrum (operation refused).	
6. Adult. Tubal discharge, attic perforation closed.	

\*Radical mastoid performed later. Healed cavity.



CHART II.—RADICAL MASTOIDS FOR C. S. O. M. WITH CHOLESTEATOMA.

Name	Age	Dura	Prev. Oper.	Labyrinth Symptoms	Intracranial Symptoms	Other Findings	Operative Findings
Gril.	37	20	—	Transient, 2 yrs. Acute attack 2 mos. previous.	Lt. temporal and occipital headaches; forgetful; depressed	Lt. 6th nerve palsy; dead labyrinth; spinal press. 270	Open fistula horizontal canal. Large exposure of dura of middle fossa.
Bro.	8	5	—	Transient	Temporal pain	Acute exacerbation; positive fistula	Large cholesteatoma cavity of entire mastoid; dura of middle and post. fossae exposed; fistula horizontal canal.
Szy.	15	3+	—	Transient	Dull temporal pain	Fistula of external auditory canal	Dura of middle fossa exposed
Rea.	27	20	2 Simple	Acute attack 2 mos. previous	Temporal headaches	Dead labyrinth; stenosis external meatus	Large cavity with destruction of horizontal canal
Sea.	21	19	2 Simple	Frequent; 1 year	Left frontal headaches	Positive fistula	Dura of middle and post. fossae exposed; fistula horizontal canal
Kap. Rt.	32	11	1 Simple	No	Unable to concentrate; generalized headaches	—	Dura of posterior fossa
Kap. Lt.	32	14	—	No	Unable to concentrate; generalized headaches	—	Negative except large cholesteatoma cavity
McD.	58	50	—	Transient, 1 year; severe 1 month ago	Lt. frontal headaches; mental confusion	Negative fistula; slight rotary nystagmus	Fistula of horizontal; facial nerve exposed over oval window
Dur.	21	4	—	No	Dull temporal	Acute exacerbation	Extradural abscess of middle and posterior fossae
Cas.	18	12	—	For 2 weeks following treatment	No	Positive fistula	No evidence of fistula; no dura exposed
Wil.	25	10	—	Transitory	Depressed; constitutional inferiority	—	Negative except cholesteatoma cavity
Cam.	44	40	—	Transitory 5 mos.	Dull temporal and occipital headaches; change in disposition	—	Dura of middle fossa exposed and covered with granulations
Qua.	22	12	—	Acute, 1 month previous	Temporal and occipital headaches	Dead labyrinth; facial palsy 3 days following removal polyp	Dura of middle fossa exposed; horizontal canal destroyed; horizontal portion of facial nerve exposed
*Maz.	6	5	Simple	No	No	No response to treatment	Aditus blocked with granulations. Cholesteatoma in antrum

NOTE.—All cases had cholesteatoma cavities.

\*Case reported as failure with treatment.

CHART III.—ANALYSIS OF CASES TREATED CONSERVATIVELY.

Name	Age	Duration Disch.	Symptoms	Treated by Others	Findings	No. Office Treat.	Results	Remarks
Bab.	37	15	Facial weakness 1 month	+ Radical advised	M. T. and outer attic wall destroyed; attic filled with C.	3	Dry 4½ yrs.	Facial weakness disappeared in one week
Cam.	40	35	Dull pain in ear 3 months	+ Radical advised	Attic blocked with C. and granulations	20	Dry 1½ yrs.	Ossiculectomy; granulations removed
Cha.	30	27	Dull pain in ear 6 months	+	Antrum filled with C. Semi-radical by nature	10	Dry 1 yr.	
Cob.	27	8	Vertigo on stooping over; dull occipital headache	+ Radical advised	Middle ear epidermatized; attic blocked with C.	3	Dry 1 yr.	Anxiety neurosis; radical mastoid operation; later with negative finding
Coo.	38	20	Fear complex	+ Radical advised	Post. sup. perforation blocked with granulations	30	Dry 3 yrs.	Radical mastoid operation refused; two recurrences
Cot.	32	2	None except discharge	+	M. T. destroyed; C. in attic; tubal discharge	40	Dry 1½ yrs.	Treated intermittently for 4 years before tubal discharge ceased
Don.	25	5	Acute exacerbation with pain	+	M. T. destroyed; granulations and C. in attic	10	Dry 6 yrs.	Three recurrences, two with otomycosis
Fri.	34	18	Pain at intervals when discharge stops	+	M. T. and outer wall of attic destroyed; large mass of C.	4	Dry 6 yrs.	One recurrence following fractured skull
Ful.	36	11	Fullness and intermittent discharge	+ Radical advised	Anterior Shrapnell perforation; attic filled with C.	20	Dry 3½ yrs.	
Gan.	30	10	Discharge	+	M. T. and ossicles nearly destroyed; much C.	10	Dry 8 yrs.	Three recurrences neglected
Gay.	28	20	Vertigo 2 weeks with decrease of discharge	+	Large polyp; post. sup. perforation with C.	20	Dry 1 yr.	Removal of polyp and ossiculectomy; one recurrence with tubal discharge
Hac.	26	20	Intermittent discharge; was dry 5 years till 6 months ago	+	M. T. destroyed; middle ear epidermatized but filled with C. Piece of old cotton in attic	4	Dry 8 yrs.	Two recurrences; one from swimming, one neglect
Hol.	58	—	Discharge for years, but dry past 10 years; fullness; tinnitus and vertigo	+	Large dry C. filling attic; Wassermann 4 plus	1	Dry 3½ yrs.	Attacks of labyrinthine vertigo, stopped following antiluetic treatment

CHART III.—ANALYSIS OF CASES TREATED CONSERVATIVELY.—Continued.

Name	Age	Duration Disch.	Symptoms	Treated by Others	Findings	No. Office Treat.	Results	Remarks
Jaq.	14	10	Giddy spells; vague headaches	+	Large perforation with destruction of malleus; granulations and C. in middle ear	6	Dry 3 yrs.	
Kav.	41	38	Discharge	+ Radical advised	Most of M. T. destroyed; granulations and C.	5	Dry 2½ yrs.	
Kes. Rt.	33	27	Discharge	+ Radical advised	M. T. and ossicles destroyed; middle ear epidermatized; attic filled with C.	2	Dry 1 yr.	
Kes. L.	33	27	Discharge	+ Radical advised	Same	6	Dry 9 mos.	
Lon.	35	20	Discharge	+ Radical advised	M. T. destroyed; middle ear partly epidermatized; granulations and C. in attic	1	Dry 2 yrs.	One recurrence
Max.	5	3	Discharge	—	M. T. destroyed; much C. in attic; tubal discharge	20	Dry 4 yrs.	Repeated recurrences from tubal discharge, headcolds
Red.	17	3	Discharge	—	Large polyp from stump of malleus; C. in attic	4	Dry 6 mos.	Polyp and malleus removed
Rlc.	48	25	Discharge	+	M. T. destroyed and outer attic wall; much C.	4	Dry 3 yrs.	
Rey.	48	40	Discharge intermittent	+	M. T. destroyed; middle ear epidermatized; C. in attic	4	Dry 6 yrs.	Two recurrences; open tube
Rog.	43	4	Discharge	+	Sharpnell perforation blocked with granulation and C.	3	Dry 8 yrs.	Stump of incus removed; two recurrences neglected
Ste.	32	17	Dizzy 10 days with pain in ear	—	M. T. destroyed; attic fistula test	10	Dry 7 yrs.	Radical operation refused
Sto.	23	20	Discharge	—	M. T. destroyed; middle ear epidermatized; C. in attic	6	Dry 2 yrs.	
Syb.	21	11	Discharge; pain and tenderness over mastoid	+	Large attic perforation blocked with C.	8	Dry 4 yrs.	Simple mastoid operation revealed C. filling antrum with granulations through aditus; C. removed and wound closed; ear dry in 3 weeks; one recurrence from swimming

Tha.	30	26	Discharge	+	Radical advised	Semi-radical by nature with epidermatization; large mass of C.	3	Dry 2 yrs.	Two recurrences with tubal discharge
Twy.	27	7	Discharge	—	—	Posterior attic perforation with C.	6	Dry 8 yrs.	
War.	28	25	Dull pain at times	+	+	Post. attic perf. and C.	4	Dry 6 yrs.	Two recurrences with head-cold
Zey.	35	30	Discharge	—	—	Complete radical by nature; cavity filled with C.	2	Dry 6 yrs.	
Bat.	60	3	Recur. disch. ear had been dry for 7 yrs. previously	+	+	M. T. destroyed; middle ear and attic filled with C.; open tube	12	Dry 9 mos.	Delayed result due to neglected and otomycosis
Ful.	36	1 wk.	Discharge; feeling of pressure; no previous history of discharge	—	—	Anterior central perforation with tubal discharge; also anterior Shrapnell perforation with C.	35	Wet	Clear tubal discharge continues; attic perforation closed after about 10 treatments; no symptoms 4 years
Hel.	33	30	Discharge	+	Radical advised	M. T. and ossicles destroyed; ulceration in tube month; much C.	25	Wet	Radical refused; tubal discharge; probably granulations in antrum
Tod.	32	26	Discharge	+	+	M. T., ossicles and outer attic wall destroyed; tubal discharge; C. in attic	45	Wet	Observation and treatment 5 years; dry 2 to 3 months at a time, but has repeated recurrences with tubal discharge
Kes.	5	3½	Discharge following scarlet fever	+	+	M. T. destroyed; attic filled with C.	12	Wet	Active tubal discharge; small particles of C. at intervals; needs radical
Maz.	8	7	Discharge; under-nourished; simple mastoid at 3 years	+	+	M. T. destroyed, also handle of malleus; attic filled with C.; tubal discharge	30	Wet	Active tubal discharge; no response to treatment; radical done; granulations in aditus
McM.	7	5	Discharge	+	+	Kidney perforation involving posterior superior quadrant, also tube; C. in attic	30	Wet	Simple mastoid done with dry ear for 5 months; repeated tubal discharge with recurrence of C.; needs radical

NOTE.—C = CHOLESTEATOMA.

CHART IV.—RECURRENCES.

	Years since ear first became dry	Number of Recurrences	Duration of Recurrences	Reason for Recurrence
1.	3	2	3 weeks	?
			3 weeks	?
2.	7	4	2 weeks } 2 weeks } 3 months } 3 months }	Head cold with tubal discharge Otomycosis Otomycosis
3.	4	1	4 months	Fractured skull
4.	6	3	4 months } 2 weeks } 4 weeks }	Neglected Head cold with tubal discharge
5.	8	2	1 month 2 months	Swimming Neglected for three years
6.	1½	1	1 month	Head cold with tubal discharge
7.	3	Several	3 weeks	Tubal discharge with every head cold
8.	7	2	3 months 3 months	? Neglected two years
9.	3	1	2 weeks	Swimming
10.	2	2	2 weeks } 2 weeks }	Tubal discharge with head cold
11.	7	2	1 week } 1 week }	Tubal discharge with head cold
12.	6	3	2 weeks } 2 months } 3 weeks }	Tubal discharge with head cold
13.	9 months	1	2 weeks	Tubal discharge with head cold

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## LXVIII.

### LYMPHOGRANULOMATOSIS AND ITS SIGNIFICANCE IN DIAGNOSIS OF DISEASES OF WALDEYER'S RING.\*

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Among a great many most complicated and so far unsolved questions regarding diseases of the blood-forming system is the problem of pseudoleukemia. The majority of works on this subject are still to be found in journals of internal medicine. This is because the hematopoietic organs are still regarded as belonging in the province of the internist and such patients are generally referred to him. Moreover, it may be that when patients of this kind come to specialists the latter do not diagnose the disease.

The clinical picture and diagnosis of these diseases make it necessary first to solve many complicated problems, and this point is most important for a specialist in ear and throat diseases, for an early diagnosis is needed so as to begin treatment at an early date and in a correct way. This proves that a detailed investigation on the subject is wanted in our special literature.

The English physician Hodgkin was the first to write on this problem (1832), describing seven cases of enlargement of the cervical lymphatic glands occurring simultaneously with enlargement of the spleen. The diagnosis given by Hodgkin was founded on clinical changes and had no scientific foundation. Therefore it could not satisfy the other investigators, for the same clinical symptoms appear in several other diseases. Between 1856 and 1866 Wilks, in England, and Wunderlich, in Germany, wrote several papers on this subject. The former recognized the priority of Hodgkin in the discovery of the disease but stated that such nodules need not always be restricted to the cervical area, but may also appear in other parts of the body and that the development of the disease brings about a cachexia and later death. This latter author noticed the resemblance this disease bore to leukemia, with the difference, however, that in the former characteristic changes in the white blood cells were lacking or were

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only very faint. These were the first observations on which later studies of pseudoleukemia were founded. In the later works we find that many authors have endeavored to discover in the histopathologic analysis an explanation of the changes in the glands, so as to be able to fix more precisely the terminology of this complicated disease. Unhappily these numerous studies did not bring the authors to an agreement. Cohnheim has, for instance, called this illness pseudo-leukemia or lympho-adenoma without leukemia. Wilks named it anemia lymphatica; Virchow, lymphosarcoma; and many more names have been suggested by others.

It was not until 1893 that Rundrath isolated an independent disease from the group and proposed to name it lymphosarcomatosis, and afterwards Paltauf isolated still another group, described by Sternberg as a special disease of the lymphatic system, of tuberculous nature.

As to the etiology of the illness many points are not clear. The fact that so many theories have been advanced and numerous micro-organisms discovered proves that none of these theories is sufficiently well founded and that the question demands further study. Ebstein and Pelle thought there must be some sort of specific provocative agent in this disease, causing chronic recidivating fever, but they could not tell what it was. One of the most promising theories, which seems well enough founded, is that of Sternberg, who connects the glandular lesions with tuberculosis because in his own eighteen cases he sometimes found at histologic examination a picture typical of tuberculous lesions, and in some few instances he could even distinguish tubercle bacilli. Some investigators (Ferrari, Caminotti and Schur and Steinhaus) accepted the explanations of Sternberg and described their own cases as a peculiar kind of tuberculosis. But there were others who expressed contrary opinions (Clarkes, Simons and Falkenheim). Still, lymphogranulomatosis must be divided into four groups with regard to its connection with tuberculosis, as has been pointed out by Clarkes. In some cases the clinical picture is identical. In the second group fall independent forms which have nothing whatever in common with tuberculosis, and in the third and fourth groups, cases in which lymphogranulomatosis has joined tuberculosis, or tuberculosis has been followed by lymphogranulomatosis.

Vamasaki describes the connection existing between lymphogranulomatosis and tuberculosis and the reciprocal dependence of the diseases on one another. This author inoculated lymphogranuloma-



tous material into experimental animals and his results led him to the following conclusions: In some of the cases, when tuberculosis bacilli were present, inoculation produced changes in the lymph tissue, but when tuberculosis bacilli were not to be found the result naturally was a negative one. Accordingly he concluded that there is no connection.

In most cases the diseased glands are enlarged, sometimes attaining the size of a hen's egg. At first the enlarged glands are soft, freely movable and painless; later, when connective tissue has penetrated into them, they become a little smaller but considerably firmer, even decidedly elastic. When a group of glands is attacked by the process, the separate glands adhere to each other, being also fixed to the deeper lying tissues, though the skin nearly always remains free. While the glands increase in size many secondary symptoms develop, caused by compression of blood vessels and nerves; the face may become edematous, exophthalmus may ensue, as well as edema of the larynx and of the trachea and bronchi; the voice becomes nasal and neuralgic pains arise. The glands are not involved in any fixed sequence, for the process spreads not only along the glandular paths but also follows the blood stream. If the initial localization is in Waldeyer's ring we observe extreme hyperplasia of the lymphatic tissue there. In our own case the palatal glands occupied the entire nasopharyngeal space, pressing on the soft palate and slightly protruding from behind the uvula. If the laryngeal glands are involved they may reach the median line, lying close to each other (D. Coopermann). Signs of involvement of Waldeyer's ring are difficulty in breathing through the nose, difficulty in swallowing, reduced hearing, tinnitus aurium and epistaxis. When the glands are involved the nasal mucosa is thickened and the nasal conchæ may be enlarged. The nasal mucous membrane is easily injured. Such patients have a mask-like face. The visible mucosa is slightly cyanotic. Later deglutition becomes difficult, speech is inaudible and the patients speak through their noses. The pharyngeal and laryngeal mucous membranes very often show hyperplasia through lack of nasal breathing. If the site of the lymphogranulomatosis is lower in the respiratory tract, the voice may change. Constant hoarseness, constriction of the pharynx, trachea and esophagus are possible (the case of Vissarewa). Among these general signs great significance is attributed to changes in the blood, for we have to deal with an unknown infectious disease influencing the hematopoietic organs. The reaction of the blood and of the hematopoietic organs is considered important for, according to the modern conception, the lymphatic system is considered a part

of the hematopoietic system. Many authors (Schur, Naegeli and Fabian) have found neutrophilic leucocytosis to be of common occurrence in this disease, though not a constant sign, for in many cases there was eosinopenia. According to the findings of Fabian, the number of leucocytes varies between hyperleucocytosis (58 per cent) and leucopenia (20 per cent), and in 32 per cent of the cases a moderate leucocytosis is found (fifteen to thirty thousands). In some instances the leucocyte count is normal. Polynuclear neutrophils are present (up to 99 per cent), and the count of leucocytes accordingly drops to 3 per cent, sometimes even to 0 per cent (W. Friese). In most cases the number of lymphocytes and monocytes is lowered. Schroeder points to this fact, for he supposes that in analyzing the blood after long intervals we ought to find a rising lymphopenia with simultaneous increase of eosinophils; nevertheless, variations are possible. The red elements of the blood may give us no changes at all or very slight alterations at the beginning of the affection, but with the progress of the disease cachexia brings about a secondary anemia with severe loss of erythrocytes (2,000,000 and less, Hg 40 = 40) 40 per cent (Boldyrew).

The temperature is most important in the clinical picture of lymphogranulomatosis. Generally it is thought that the temperature curve in lymphogranulomatosis must be a typical one, periods of high fever alternating with periods of normal temperature. Still there are cases when the temperature curve is irregular, and there are other cases which never have any fever. This is why, when the greatest attention is being paid to temperature, mistakes may be made in diagnosis. There are long lasting attacks of sweating at night and great losses in weight, causing cachexia and death. No less is the importance attributed to the analysis of the urine for urobiline and a positive diazo reaction, which is thought to be characteristic of lymphogranulomatosis (Kalantarowa). Itching is thought to be an early dermal symptom; sometimes it is most annoying. Moreover, there are frequently present skin affections, such as eczema and dermatitis. Some authors (Karnitzky) noted severe diarrhea in their cases. The examination of the inner organs often proved that the spleen and liver are involved; these organs are enlarged.

Generally the disease has a slow evolution—two to two and one-half years—although in some few cases it lasted eight to ten years or even more. There have been observed cases in which the disease lasted only from two to three months and less (Naegeli). Two stages are distinguished according to the clinical evolution of the

affection: the first is a local one, sometimes lasting several years, but generally one to two years; the general condition of the patient is rather a good one at this stage. The second stage is one of generalization in which the patient rapidly loses weight, presents signs of severe anemia and cachexia, which always lead to death. Men are found suffering from this disease two to three times as often as women. Generally the patients had formerly been quite well (Skolow, Fabian). Most of the patients are middle aged: twenty to forty years old though sometimes exceptions to this rule occur. Prissel saw this disease in a child of three and one-half months. Skwortzow, Kasantzew and Kritschewsky saw it in children aged eight to twelve years, as well as in elderly people, though this but rarely. An early diagnosis of lesions of Waldeyer's ring is most difficult, for similar symptoms are met with in several other diseases. Even later the clinical development is so complex that prolonged, painstaking clinical observations and microscopic analyses are necessary to make a diagnosis. Therefore lymphogranulomatosis of the upper respiratory tract must be differentiated from several other affections of the pharynx: infectious diseases or benign and malignant neoplasms (of inflammatory origin).

To the former group belong tuberculosis and syphilis, which are most often localized in the palatal glands and the tonsils. In cases of tuberculosis the tonsils often do not present any changes, but in the ulcerative forms a histopathologic analysis shows the character of the disease. In such cases there are also specific foci in the lungs or in other organs. In cases of local lesions prognosis is generally favorable.

Syphilitic lesions are considerably more characteristic. During the first period the tonsils are hard and severely inflamed. In most cases the affection is unilateral. The submaxillary glands and those at the angle of the lower jaw are swollen and painful. There is general malaise, deglutition is painful and there is fever. Later ulcerations as well as a diphtheroid thin coating appear on the tonsils. When the secondary signs, such as papulous rashes, have developed and the Wassermann proves positive, the true nature of the disease may be ascertained. If treatment is begun in due time and conducted in a regular way success is generally attained.

If we compare lymphogranulomatosis to the above mentioned infectious granulomas we note that there are many clinical signs pointing to lymphogranulomatosis, for in most cases the other kinds of granulomas cause no alterations in the neighboring organs and

tissues, and there are no ulcerations at the site of the tumor. Moreover, in the later development of syphilis there are typical secondary symptoms, such as papulous rashes on the skin, and in tuberculosis the ulcer grows larger, invading the surrounding mucous membrane. In cases of syphilis we find a painful scleradenitis. But this is not the case in lymphogranulomatosis, for at the beginning of the disease in Waldeyer's ring the neighboring glands may be free or only slightly enlarged but soft and not painful. In cases of tuberculosis the cervical glands may be free or show changes typical for tuberculous adenitis. Where treatment has begun early and been thorough, the syphilitic symptoms may disappear completely and tuberculosis may develop a benign course, which is never the case in lymphogranulomatosis, for here the process develops notwithstanding the therapeutic measures taken and death is sure.

Judging by the appearance and by the clinical development of the disease, it is comparatively easy to exclude the existence of benign neoplasms, such as lipomas, choanal polyps, hemangiomas and teratomas. It is more difficult to differentiate between lymphogranulomatosis and malignant growths; melanosarcoma, carcinoma, lymphosarcoma, endothelioma, fibroma, lympho-epithelial growths of Schmincke's type and lymphosarcomatosis (Rundrath).

They have each some characteristic peculiarities by which they are distinguished from one another, but it is very easy to mistake them for lymphogranulomatosis. As has been stated before, blood symptoms are not always characteristic in such cases. Considerably greater is the importance of the intermittent fever of the type Pelle-Ebstein, but this too is often lacking. Analysis of the urine and diazo reaction are not always helpful, for they may yield negative results in many typical cases. Of great import for diagnosis is the simultaneous enlargement of the spleen and liver, which protrude considerably from under the ribs. Still greater is the diagnostic bearing of typical fever, accompanied by painful itching, but unfortunately this is likewise no constant symptom. In comparing the above malignant neoplasms of the same localization with lymphogranuloma it must be said that in the case of the latter the pharyngeal glands are hardly ever ulcerated; this distinguishes them from sarcoma, carcinoma and lympho-epithelial growths of Schmincke's type. Moreover, in instances of local involvement of superficial glands the latter are not painful. But if cervical glands are attacked there is no special order in which the symptoms appear, while in cases of malignant growths metastases first of all occur in submaxillary and cervical glands. In lymphogranuloma too the glands are rather soft

and painless at the beginning, moving freely and only get firmer in the course of further progress, which is not the case in carcinoma, for there the glands may at first be very firm and even more enlarged than later. The glands coalesce and adhere to the lower-lying tissues, whereas they are never fixed to the skin and do not adhere to it, as is the case with lymphosarcoma. Such neoplasms as fibromas and melanosarcomas may easily be excluded by their appearance and evolution. The former are localized only in the nasopharyngeal space and in the nasal cavity, being firmer and nearly cartilaginous. They have no metastases. The glands are not involved. The latter are of a typical brownish-dark color. Such neoplasms as endotheliomas may also easily be excluded, for they rarely involve the neighboring glands and have no metastases. They show a tendency to local recurrence and do not cause cachexia. Therefore it is only difficult to distinguish between lymphosarcoma, sarcoma, lympho-epitheliomatous growths of Schmincke's type and lymphogranuloma. But this is of no great consequence for, according to Pissarewa, Genatzi and other authors, lymphogranuloma in the early stages of its development may be a circumscribed process; sometimes the disease only appears in the shape of local lesions. Early operation and roentgen ray treatment give best results in the initial periods of all three affections. As to the problem of diagnosis, it is best solved by means of biopsy. This sometimes yields definite results only when repeated several times. After all these considerations of a general kind we shall now describe our own case, which is most illustrative, being difficult to diagnose and considering the effectiveness of surgical treatment.

#### REPORT OF A CASE.

CASE 1.—K. O., aged 52, farmer's wife, was seen on April 28, 1933, complaining of impossibility of nasal breathing, giddiness, nose bleeding, intense itching of the skin; the patient stated that she tired very easily. The patient has hardly ever been ill. She stated that there had never been any tuberculosis or syphilis in her family. She had been married for thirty years. She is childless. The present illness she noticed about four months ago. Two months later nose breathing became quite impossible. During only the last fortnight she suffered from headaches and nose bleeding with itching of the skin. The patient is of medium height and normal build. The subcutaneous fat tissue is normally developed, but the chest and back are covered with scratches. The nasolabial folds are slightly effaced. The lips are bluish. The glands of the buccal cavity, as well as cervical, supra- and infra-clavicular glands are not palpable. There are no changes in the inner organs. The mucous membrane is hyperemic.

The soft palate and uvula are lowered; in phonation only slightly mobile. The mucous membrane of the posterior wall of the glottis is pale and a little dry. The tonsils are deeply buried. The whole of the nasopharyngeal space is filled by

a neoplasm of dough-like consistence, which is visible behind the uvula. Digital palpation shows that the tumor is fixed to the superior posterior wall of the nasopharynx by a broad base. It is impossible to palpate the tumor from all sides, as its posterior part is filling the choana. The tumor is of rounded shape, covered with a shining reddish capsule, on which some dilated capillaries are visible.

Uniform hyperemia of the mucosa of the pharynx. Both tympanic membranes are dulled and slightly retracted. The roentgen ray examination of the adnexa of the nasal cavities, of the cervical vertebrae and lungs did not reveal any changes (F. Podolsky). Wassermann reaction and Pirquet were negative. Blood: Hb. 95 per cent, erythrocytes 5,100,000, leucocytes 6,800, Col. Ind. 0.9, eosinophiles 26 per cent, rods 5 per cent, segmented 25 per cent, lymphocytes 40 per cent, monocytes 1 per cent. Türk's cells 1 per cent; anisocytosis, poikilocytosis (P. Tihanowsky). Urine: 100 cm. of straw-yellow, slightly troubled urine; acid reaction; specific weight, 1013; slight sedimentation; 66 per cent; sugar, gall pigments, acetone, indican, urobiline and blood corpuscles are lacking. Diazo reaction is negative. Casts: One to two in a visual field. No erythrocytes. Leucocytes, five to six in a field. In the sediment there are amorphous urates and oxalic crystals (Tihanowsky).

May 6. Operation under superficial anesthesia, 10 per cent sol. cocain. Patient's head in Rose's position. The soft palate is drawn forward with a blunt hook. The neoplasm filling the entire nasopharynx is cut with a pair of Cooper's long scissors under control of a finger. Bleeding is slight. Nose breathing is sufficient. Up to May 18th, the patient daily instilled five drops of a 3 per cent protargol solution in each nostril twice a day. The temperature was normal all the time. The only unpleasant sensation was caused by itching and diarrhea, but these untoward symptoms receded under influence of local therapy.

May 18. The rests of the tumor of the nasopharynx were excised with an adenotome.

May 20. The patient left the hospital, her nasal breathing being completely restored. Later the patient was treated at the clinic and this afforded us the opportunity to observe her for two months longer. During this time she suffered from intermittent attacks of diarrhea and itching and the tumor did not recur. Later we advised the patient to go to a roentgen institute for treatment. We heard of her last in December, 1933. She wrote to say that the results of the treatment she had received were quite satisfactory. She was able to do her work, and during these seven months there was no recurrence.

The microscopic analysis of the excised tumor revealed the presence of lymphogranulomatosis of a hyperplastic, lympho-endothelial type, with considerable eosinophilia (Beradjansky).

In examining the diseased glands of Waldeyer's ring with the naked eye we noticed that some glands were greatly enlarged, whereas others remained of normal size. Such an enlarged gland was sometimes as big as a pigeon's egg or even bigger. Such an appearance was seen at the beginning of the illness, but in later periods, when the process had advanced, we found granules at the posterior walls of the glottis and on the lateral ridges, and the tonsils are also involved. On examination with the naked eye we saw that the appearance of

the diseased gland depended on the stage of the development of the pathologic process. At the beginning the gland is slightly enlarged, has a rounded shape, being of uniform, dough-like consistency with a smooth, slightly distended capsule, showing at the surface capillary vessels which are also a little distended and cyanotic. On the section, the tumor resembles a neoplasm of inflammatory origin with the difference that there are layers of whitish, thread-like substance dividing the areas of uniformly colored glandular tissue from each other and lending the cut the look of marble. In some instances there are areas of a brighter coloring among the uniform hue of the infiltrate. Such bright spots are no greater than a pinhead. Later the color of the gland changes from bright red to pale pink; sometimes it even turns grayish yellow. The capsule of the gland, hitherto shining and transparent, turns dull and dry. On the surface of such an enlarged tonsil there are petechial bleedings. As the process develops the tonsils become much firmer. When they are cut strands of connective tissue are visible at the section, and in some parts elements of fibrous tissue appear, among which there are necrotic areas. Generally such necrosis is distributed in an irregular way. Among the internal organs the spleen and liver are involved most often, especially the spleen. Its surface is then uneven, firm and rough. On section it shows tubercle-like formations of different size and shape. The color of the spleen is variable, from a grayish yellow hue to brick-red color. The nodules are rather firm, but the rest of the spleen is considerably softer. Owing to such a checkered appearance of the spleen, it has been designated as porphry spleen. In most instances the liver is not enlarged. It is pale and there are whitish nodules disseminated in it, some being as large as hazelnuts, others even larger. The histologic examination showed a considerably more complicated appearance, for the kind of alteration is in a great measure dependent on the stage of the process. In the beginning we find an appearance most typical of inflammatory infiltration. This resemblance is caused by a great number of lymphocytes, plasma cells and polynuclears mixed together in various proportions. The histologic appearance of the gland has little in common with the typical structure of the same. Characteristic of this condition is the presence of many polynuclears, often also of eosinophiles, amongst which there are more or less important accumulations of giant cells, polynuclears, plasma and epithelial cells. The finding of peculiar giant cells by Sternberg caused them to be considered as a constant symptom in lymphogranuloma, but lately there have been well established cases of this disease without any giant cells. At this stage of development



the microscopic findings often lead the observer to mistake it for sarcomatous neoplasms. As the process progresses the number of different kinds of corpuscular elements diminishes, the lymphoid tissue is replaced by fibrous tissue which later undergoes hyalin degeneration, and in some areas of lymphoid tissue, which is preserved though greatly altered, there appear foci of necrosis which suggest a caseous focus of tuberculosis, whereas it differs from this by the absence of calcified deposits. An especially weighty significance is attributed in lymphogranulomatosis to the existence of vessels which allow us to distinguish the same from tuberculomas. Later, under influence of pressure, the lumen of the vessels is filled up, the surrounding tissue suffers in its nutrition and this causes dissolution and necrosis. As to the other organs, it must be noted that such infiltrations occur in all tissues of the human organism and sometimes even in places where lymphoid tissue is lacking. The liver may serve as an example. When analyzing the liver tissue histologically we find there such formations. The nodules are of very variable structure: they consist of differently shaped connective tissue cells of different volume. Sternberg's giant cells lie freely among these cells. In the spleen too connective tissue strands are noted; they change the typical appearance of the spleen tissue so as to be hardly recognizable, but in the small areas of intact tissue we find the same kind of cells as in lesions of the lymphatic glands. Although there are diverse microscopic signs characteristic for lymphogranulomatosis, this disease still lacks a typical classical aspect. Therefore even now mistakes in diagnosis are possible if histologic findings are the only basis of the diagnosis. Owing to our faulty diagnostics and our lack of understanding of the etiology of this disease, therapy is as yet unsuccessful. Therefore, to our great regret, the treatment is still directed not against the principal disorder and the success obtained by our measures is but of temporary kind.

All methods of treatment that have been submitted may be divided into four groups: treatment by medicaments only, combined method of using medicaments and roentgenization of the diseased tissues, roentgen ray and radiotherapy and surgical treatment combined with roentgen therapy. Of the medicinal substances in use arsenic is given in small as well as in large doses, iodides, salvarsan and many antiphlogistic drugs, and various narcotics are recommended. Of course, none of the drugs has as yet proved helpful. If there have been cases of healing they ought rather to be regarded as cases of granuloma, of specific etiology (Naegeli). Much better results have been due to X-ray therapy, which proved



most useful in many instances: the glands became smaller; sometimes they even disappeared entirely. Generally the success of roentgen ray therapy consists in the fact that under its influence the hyperplastic lymphoid tissue is replaced by specific scar tissue, which may later bring about hyalinization of the gland. In several cases this method of treatment prolonged life for three or four years, and it even happened that seven to ten years passed without recurrence (Goldstein and Zuckermann). On the contrary, it has also been observed that roentgen therapy did not cause any improvement of the condition, which grew worse. This was due to the fact that roentgen therapy demands the use of exact doses, the success of the cure depending largely on this. The majority of authors hold that in most cases there is improvement after several sittings of ray therapy; the improvement may be either general or local. This way of treatment is most important if the lesion is in the upper respiratory tract, for if the infiltration greatly recedes in size this may save the patient from many dangerous complications. Lately radiotherapy has yielded even better results. Davids has seen among five cases and Berneumont among one hundred and seventy-three cases much better results than those obtained by roentgen therapy. Still we must remember that the results of roentgen therapy are never definite; that only a resorption of the infiltration is caused but the disease is bound to recur sooner or later, killing the patient. No less is the significance of surgical treatment combined with ray therapy. T. Pissarewa and S. Geinatz pay special attention to this kind of therapy. Basing their view on their own observations and on cases of other authors (Hertzen, Stchiogolew and Bibel), they think that in cases of early diagnosis, when the position of the lesion permits operation, surgical measures are indicated, for if they are followed by roentgen therapy the results are good. Especially great is the significance of such a mode of treatment in otolaryngology, where early diagnosis often proves possible, not only thanks to visibility but also thanks to biopsy. Surgical operation is indicated if the growth is localized in the nasopharynx, for two reasons: First of all it is possible to excise the primary growth as has been pointed out by some authors (Goloobow). If we consider that some facts allow us to regard lymphogranulomatosis as an infectious disease we must agree that it demands operative treatment. In many diseases of the inner organs we excise the tonsils if we have reason to suppose that infection starting from the tonsils is causing intoxication of the organism. Moreover, we make nasal breathing possible when we clear the nasopharynx of the hyperplastic lymphatic tissues, and later roentgen

ray may be used in the treatment of the remainder of the diseased tissue. Roentgen therapy alone cannot give such results. In cases where the palatal glands are involved it is quite possible to enucleate them completely and to cause complete resorption of the lymphogranulomatosis by applying roentgen therapy. No less important are the reasons for treating lymphogranulomatosis by operating on it when localized in the upper respiratory tracts, if we agree that it has much in common with malignant growths. We know that in such instances we may be able, if not to save the patient, still to produce a lasting effect. Of course this regards cases with local lesions in which the general condition is a good one, and if there is reason to assume that there are no general symptoms as yet. Notwithstanding all these beneficial results, some still think operative treatment useless, and there even are some who think it harmful. Still operative treatment, followed by roentgen ray therapy, remains the only method which gives, if not complete recovery, still improvement of the condition in cases with strict indication. It prolongs the life of the patients and renders them capable of working. Therefore we have reason not to proclaim ourselves beaten, not to regard such patients as lost, but to make use of this method of therapy, though it may not be a radical one, for in combination with roentgen rays it allows us to keep off cachexia for a time and therefore also death.

#### CONCLUSIONS.

We may conclude that:

1. There is cause at the present state of knowledge to suppose that in cases of early diagnosis local circumscribed form of lymphogranulomatosis may be found.

2. In many instances it is most difficult to differentiate lymphogranulomatosis of Waldeyer's ring from malignant tumors; therefore biopsy is indicated.

3. Operative treatment combined with roentgen therapy is at the present state of knowledge the most rational one.

13 2ND SOVIET STREET.

# Clinical Notes and New Instruments.

## LXIX.

### TWO CASES OF MASTOIDITIS FOLLOWING THE USE OF VERY HOT SOLUTIONS IN THE EAR.

WILLIAM GILLIAM KENNON, M. D.,  
NASHVILLE, TENN.

#### REPORT OF CASES.

CASE 1.—Mrs. C. B. P., age 26, white whose previous history had no bearing on the condition for which she consulted me, was first seen on July 25, 1933. The history was that on the night before she thought she had an insect in her right ear. Her husband heated some olive oil in a teaspoon and poured it into the ear. The oil was quite hot, according to the patient's statement, and there was what she described as agonizing pain for several minutes. She came to my office the next morning, not particularly on account of pain, but to have the bug, which she thought was still in her ear, removed. Examination of the right ear showed no foreign body. The drum membrane was slightly pink and there was a small bleb on the central portion of this structure. The left ear was normal in appearance. I did not think there were any particularly serious results to be anticipated and so informed the patient.

Four days later there were noted an excoriation on right external auditory canal wall and a small perforation of the drum membrane in the anterior superior quadrant.

Two days later the anterior half of the drum membrane had disappeared. There was a profuse purulent discharge from the middle ear.

On August 21, 1933, there had been definite mastoid tenderness for past few days and the profuse discharge continued. X-ray showed clouding of entire right mastoid. August 25, 1933, X-ray was repeated, with essentially the same findings.

August 26, 1933, mastoid operation was done. All cells were found to be full of pus and edematous mucous membrane; culture from the pus showed streptococci.

CASE 2.—Mrs. H. A. H., age 22, white, complained of pain in the left ear for the past fourteen hours. The ear trouble followed an attack of acute tonsillitis. The left drum membrane was red and there was a small bleb on the posterior superior quadrant. There was marked inflammation of the tonsils and the pharynx. A prescription for 5 per cent phenol in glycerine was given.

Two days later the patient stated that she had not been entirely free from pain at any time since my first visit. The ear at this time showed a bulging, red, drum membrane with all the other symptoms of an acute otitis media. The patient now confessed that some very hot drops had been introduced by a neigh-

bor before my first visit. The pain at the time was described as being agonizing. I opened the ear drum on March 3, 1934, under nitrous oxide anesthesia.

March 12, 1934, there was a profuse discharge from ear since myringotomy, and considerable pain for three days following this procedure. Profuse yellowish purulent discharge filled the entire canal. No pulsation was seen after the canal had been cleared of discharge. There was slight tenderness over the mastoid tip and the antrum. The patient had no fever, slept well and had a good appetite.

March 15, 1934. Tenderness over the mastoid persisted. The discharge was very profuse.

March 20, 1934. X-ray showed definite clouding and some bone destruction of the left mastoid.

March 22, 1934. Mastoidectomy was done. Almost all cells found were full of pus and edematous mucous membrane. Culture from the pus showed pneumococci.

In the case of the second patient there is some doubt as to whether or not the very hot solution was the cause of the otitis, as the pain began before the solution was used.

528 DOCTORS' BUILDING.

LXX.

COLLECTION OF DEEP SEATED CULTURE MATERIAL  
FROM EAR, NOSE AND THROAT.

C. D. VAN WAGENEN, M. D.,  
NEW YORK.

The unprotected swab has failed as the collector-conveyor of culture material from all deep seated accessible nasal areas. Various protectors have been devised, themselves more bulky and impossible of introduction, forgetful that swab capillarity operates immediately beyond the zone of protection.

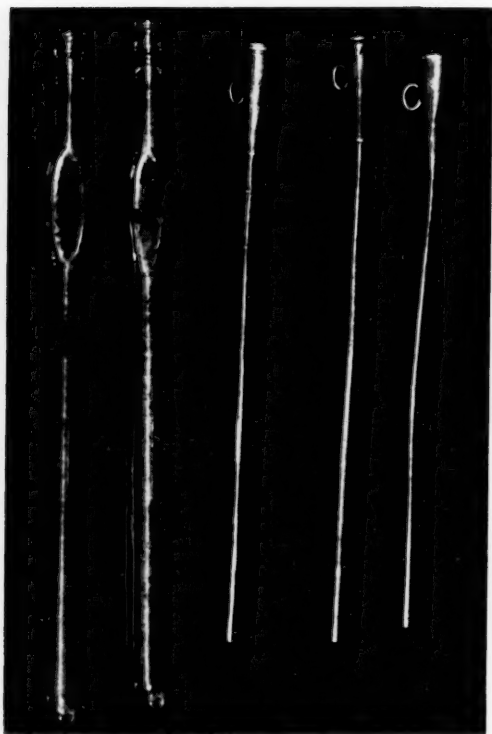
A collector is required which can be introduced, under local anesthesia, to the desired area, no matter what may be the obstruction in its path, can collect that which we wish to investigate and may be withdrawn without contamination.

For many years the writer and his pathologist have derived satisfactory results from the use of suction through malleable silver eustachian catheters and hollow glass tubes, as collectors and platinum loops as conveyors.

Two requisites should govern the selection of the suction apparatus, i. e., wide range of control and quiet action.

Controllability in the ear is important, particularly in a chronic suppurative middle ear or a radical mastoidectomy wound where too violent suction may induce a temporary vertigo and alarm the patient needlessly. It is equally important in the presence of an acute accessory sinus inflammation and in the handling of apprehensive or rebellious patients. Two types of suction instruments, aside from some form of hand bulb, meet these requirements, i. e., the Connell anesthesia apparatus and, less convenient, an attachment to the water faucet. Most other suction pumps are too noisy, have but one speed and are too violent in action.

Collection in acute middle ear suppuration is easily and satisfactorily made through the myringotome as collector-conveyor, a separate knife being used if the infection be bilateral. Otherwise a straightened eustachian catheter, armed with a rubber bulb which may be compressed during introduction, may be passed up to or



through a perforation in the membrana tympani, bulb compression released and the catheter withdrawn. Its interior will be coated or filled with the latest withdrawal from parts beyond the perforation. We are not concerned with what may become adherent to the outer surface of the catheter save that we must avoid touching this outer surface during the next step.

The flamed platinum loop is passed into the lumen of the catheter and rotated between thumb and finger to coat it with the contents of the catheter. The loop is withdrawn and the appropriate medium is impregnated. The withdrawn loop must touch nothing but the medium. If it does, the loop must be resterilized and another attempt made. The cork and the rim of the tube must be held in the flame before recorking. If the medium be a plate, it must be recovered immediately after streaking. In the open mastoidectomy wound preliminary sponging should be avoided as far as possible, and the loop plunged into the visible pus.

Few intranasal obstructions will be encountered which may not be circumvented by the malleable eustachian catheter. A preliminary cleaning of each naris should be done, with a special catheter and suction, and the catheter discarded immediately. This step has the twofold advantage of removing irrelevant material and establishing a certain degree of tolerance in the patient. A second catheter, bent to the required shape, is then passed into the area under investigation and the current turned on. Suction is stopped before the catheter is withdrawn. If the discharge is tenacious, momentary compression of the nares will promote flow from the deep area.

667 MADISON AVE.



# The Scientific Papers of the American Bronchoscopic Society.

LXXI.

ADDRESS OF THE PRESIDENT.\*

WAITMAN F. ZINN, M. D.,  
BALTIMORE.

For several years it has been my great pleasure and privilege to serve as an officer of this Society. I wish to express to you my deep appreciation of the honor you conferred on me at our last meeting in electing me to fill this, the highest office—that of President of the Society. I wish to say that much of the credit for the work that has been done must go to Dr. Clerf, who kindly consented to act as secretary for us this year. My sincere thanks go to him for his thoughtfulness and co-operation.

The rapid progress that the American Bronchoscopic Society has made during the past few years in stimulating the interest and incentive within its membership was most forcefully brought to my attention recently when Dr. Clerf reported, concerning the program for this meeting, that we were exceptionally well supplied with an abundance of valuable and interesting papers to be presented to you today. Our problem, then, is no longer one of a dearth of material, as in the early days, but rather, as it should rightly be, a question of time in which to bring before the assembled membership the experiences and discussions of interest to you all. With this thought in mind I shall limit my own remarks to a brief survey of the historic mileposts in the development of endoscopy.

I have in my possession a most interesting volume, copies of which some of you may have seen, although I have been told that the book I have is the only one in this country. It has the ancient lengthy title: "*Some Account of Lord Boringdon's Accident on 21st July, 1817, and its Consequences.*" It recounts in detail the story of the accidental aspiration of an ear of rye by an eleven-year-old boy, the symptoms produced and the treatment administered. Compare the methods of the group of distinguished English physicians who

\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

labored to save the life of this young nobleman with similar cases as you see them in your clinics today.

The initial choking lasted some five or six minutes, after which no inconvenience was felt. Goodly quantities of bread were given at once and swallowed without difficulty. It was thought that the corn had been coughed up or had passed into the stomach. The quiescent state of the lungs on the days immediately following the accident led the six doctors assembled from Paris and London to concur in the opinion that the passage of the ear of rye into the lungs was an impossibility, to which they were unwarranted in referring the symptoms of the case. On the seventh day the child had a hemorrhage from the lung. He was bled, and leeches were applied at about weekly intervals during the remainder of the summer. "Plaisters" were frequently used. It was found that when he was carried about the garden in a chair balanced between two poles, the motion had a powerful effect in lowering his pulse.

It was not until October 1, about two and one-half months after the accident, that he first complained of pain in his side. Leeches gave immediate relief. From this time until his death on November 1, he had increasing pain, diminution of appetite and general malaise. Opium was given to allay the debilitating cough. Several times when he was particularly low, five, instead of the customary four glasses of wine were administered, and the patient rallied. Twice the doctors attempted to operate with a view to draw off the "matter" which had collected from the abscess, which they located as near the liver. It was thought the material had oozed into the chest and was producing the subsequent difficult breathing. It was found that adhesions made drainage impossible. The following evening the child died, "such an instantaneous death that his lungs were found distended with air; a small quantity of matter entering the large blood-vessel occasioned the heart to stop."

The account goes on to say that even had the attending medical gentlemen been able to see into the child's body and to have located the exact position of the ear of rye, they would have been powerless to have afforded him any relief. Their judicious treatment alleviated the different symptoms as they arose, and they were encouraged by hope. Further insight into the situation would have been but "melancholy knowledge occasioning exertions more laborious and anxieties more painful."

It is needless for me to point out to you who know it so well, the course such a case might follow today. This child had the misfortune to experience this distressing accident long before necessity and medical science had given to the doctor the ability to look directly

into the lungs and esophagus. During the boy's illness his father had researches made in England for analogous cases. Much industry finally revealed that in 1662 a young college student had aspirated an ear of barley which produced fever, cough, spitting of blood and pain in the right side where an abscess localized and was incised, emitting the green barley head quite unchanged. The patient recovered. In 1703 at Frederickstadt, a twelve-year-old boy had a similar accident, the barley ear again being extracted from the localized abscess. This child likewise recovered, as did a Swiss boy, aged eleven years, who, from the symptoms produced by the aspiration of a bit of straw—in the year 1795—was deemed a consumptive with no hope of recovery. This child managed to cough up the foreign material and gained immediate relief. As can be seen, external surgery in such cases was resorted to only at the periphery of the lung and after an abscess had formed. While the germ of the idea, which later developed into bronchoscopy, had, even at this early date (1818), stirred in the minds of a few, it was still impractical and too undeveloped to aid those who were as unfortunate as the child whose case I have just presented.

It is generally conceded, I believe, that to Gustav Killian belongs the title "The Father of Bronchoscopy." It was he who first successfully removed a foreign body from a bronchus in 1897, and he it was who developed both upper and lower bronchoscopy. However, there were numerous attempts made throughout the ages for many years prior to Killian's success, and it is these early trials which are of particular interest in that they show the struggle of an idea toward reality.

It is recorded that in 1743 the first laryngoscopic examination was made by Levret, who used a plate of polished metal instead of a mirror, but that he did not obtain very good results. Philip Bozini, of Frankfort, designed his famous apparatus for the examination of the upper end of the esophagus in 1807. The size of the instrument and the violent reflexes it produced rendered it practically unusable. Eighteen years later, John D. Fisher, of Boston, invented an endoscope using a candle for illumination. In 1827, Senne used, to examine the throat of a child, a small mirror which was the prototype of the present laryngeal mirror. Within the decade following 1829, Babbington, Bennati and Liston all attempted examination of the vocal cords with a similar type of mirror. However, it was not until Garcia, in 1856, invented the laryngoscope with which he viewed his own vocal cords, that any real progress was made. It was Turck and Czermack who perfected this method and are by some considered its true authors. At best, the examinations up to this time shed light only on the hypopharynx and glottis.

It was about 1865 that the first attempt at esophagoscopy was made. Cruise endeavored to use a Desormeaux urethroscope for this examination but his results were negligible. The following year Semelder and Stoerck, of Vienna, using this same instrument made a successful examination of the esophagus and published the results of their investigations. Kussmaul, in 1868, perfected this instrument, changing the lighting. Using it he diagnosed a cancerous tumor situated at the level of the bifurcation of the bronchus. Kussmaul used a straight, rigid tube and placed his patient in the so-called "sword swallowing" position. In the same year (1868), Bevan, in England, described an esophagoscope of his invention. It consisted of a tube four inches long and three-quarters of an inch in diameter. This tube contained a second tube fitting into it like a telescope. At the upper part of the external tube was a mirror used, with reflected light, to examine the deep parts.

There were various attempts, successes and failures, from this time until 1880, when Mackenzie, in London, took up the study of esophagoscopy. He constructed an apparatus on the principle of Bevan's, with which he was not familiar at the time. A skeleton tube was formed of two wires joined by rings placed at a certain distance from each other. It was possible to shift the rings from a vertical position, in which they were introduced, to a horizontal position while in situ, thus increasing the diameter of the tube. A reflecting mirror was fixed at the end of the tube. Reports include examination of fifty patients with successes in thirty-seven cases. Removal of a cancer the size of a cherry from a patient 62 years old, the removal of a mucous polyp of the esophagus from a woman 27 years old and removal of a fragment of bone are included in the report.

Up to this time the greatest difficulties presenting themselves were the lack of sufficient and direct lighting, and the inability to control the reflexes produced by the introduction of the tube. The invention of the incandescent lamp by Edison in 1878 was a great boon to endoscopic research. Leiter and von Hacker perfected the electric light and the reflecting system. In the meantime, Mikulicz advanced the idea of an injection of morphin to control the reflexes, and a little later (1891) Gottstein advocated cocain anesthesia. Prior to this time general anesthesia or morphin had been used.

The name of Kirstein cannot be omitted in any survey of this field. In 1895 he described, under the name "Autoscopy," a direct laryngoscopy and tracheoscopy, using an electric head light and a spatula inserted to the base of the tongue between it and the epiglottis. By pressure forward the epiglottis was drawn up, making direct inspec-

tion of the interior of the larynx possible in many though not all cases. This was the greatest advance in the examination of the larynx and trachea up to this time.

Kirstein was followed by the great Killian, of Freiberg, who improved the instruments and technic of his predecessors and astounded the medical world by his removal of a foreign body from the bronchus. His two associates, von Eicken, inventor of extracting forceps, and Brünings, designer of lamp, tubes and forceps, hold their own places of importance beside their master.

Esophagoscopy preceded bronchoscopy by many years. However, it was only with the advent of Killian bronchoscopy that the development of a special technic in esophagoscopy received great impetus and became practical and feasible. From this time on, the two went hand in hand. The rapid strides made in esophagoscopy in its later stages are due to the laryngologists, for while this work remained in the hands of isolated internists it was of little importance. Esophagoscopy has been greatly influenced by the development of the direct examination of the air passages, which progress has been comparatively swift.

The first bronchoscopy in this country was done in Boston, at the Massachusetts General Hospital, by Coolidge, in 1898. A part of a tracheotomic cannula was removed from the right bronchus of a young man of twenty-two. Rapid progress has been made since the advent of the twentieth century. In 1902 Einhorn devised an esophagoscope with an auxiliary tube within the wall of the main tube. A light carrier was inserted in the auxiliary tube to carry light to the distal end of the tube. Guisez and Ingals reported removal of a nail and a pin, respectively, at about this time.

Killian's place at the head of bronchoscopic research abroad is filled in this country by our own Chevalier Jackson. He it is who gave us, in 1904, an instrument combining the best features of the earlier models of Einhorn and Killian, and, a year later, one having an auxiliary canal and a drainage canal. His continuous study of our problems, his writings, his organization of this Society and his work as first President are well known to us all.

There are many whose names have been necessarily omitted in such a brief summary. To go into the list of those who are today prominent in our field would be, we are glad to say, quite an exhausting task. It is a pleasure and a satisfaction to contemplate the remarkable development of the direct methods of examination of the food and air passages. Not only has laryngology progressed but the entire field of medical science has been influenced by this research.

MEDICAL ARTS BLDG.

## LXXII.

### MINUTE PERFORATION OF THE CERVICAL ESOPHAGUS; FULMINATING DESCENDING INFECTION; MEDIAS- TINITIS; EXTERNAL OPERATION; RECOVERY: CASE REPORT.\*

CLYDE A. HEATLY, M. D.,  
ROCHESTER, N. Y.

Perforations of the cervical esophagus frequently result in a fatal extension of infection along the fascial planes to the mediastinum. The successful management of such perforations involves the correct differentiation between those cases in which localization may reasonably be expected and those in which downward extension must be feared. The well known difficulties of exact diagnosis in this respect make an external operation indicated whenever doubt arises concerning the safety of the patient, because this not only serves to evacuate extravasated material but also, if instituted early, effectively blocks the fascial pathways to the mediastinum.

The following case report illustrates the successful management by external drainage of a virulent infection carried into the neck and superior mediastinum through a minute perforation of the cervical esophagus. Its fulminating character differs from the usual insidious course so frequently encountered in cases of minute perforation. The X-ray studies illustrating the progress and control of the mediastinal infection are of considerable interest.

#### REPORT OF A CASE.

CASE 1.—F. C., a woman of 21 years, came to my office on December 16, 1933, with the complaint that she had swallowed some sharp foreign object while eating spaghetti three hours before. Dysphagia was present together with a sharp, sticking sensation along the right side of the neck at the cricoid level. There was definite tenderness on lateral pressure at this point. The patient refused advice to go to the hospital for immediate esophagoscopy. Five hours later, however, she voluntarily entered the hospital because of the rapidly increasing severity of her symptoms. The pain had become intense and swallowing practically impossible. Nausea associated with scanty and at times blood-tinged expectoration was noted. There was slight

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\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

From the Department of Surgery, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.

fever ( $37.5^{\circ}$  C.). X-ray studies were entirely negative. Immediate esophagoscopy was done with local anesthesia. Through the speculum a fragment of toothpick one inch in length could be seen, in the upper esophagus in the antero-posterior plane, with its point buried in the right posterior wall. It was removed without difficulty. There was no gross evidence of local injury about the point of lodgment. The following morning the patient appeared very ill, complaining of severe pain the neck and upper chest and complete inability to swallow. There was considerable tenderness along the anterior border of both sterno-cleido-mastoid muscles and on the right side definite crepitation could be detected at the cricoid level. Temperature  $38.8^{\circ}$  C., pulse 104, respirations 22, W. B. C. 29,000. An X-ray examination showed no evidence of mediastinal changes. The clinical picture, however, indicated a rapidly spreading infection and external drainage was considered imperative. This was done under local anesthesia following the technic previously described to this Society. An incision was made along the anterior border of the sterno-cleido-mastoid muscle on the right side and the muscle exposed and retracted. The thyroid gland was then mobilized by ligating and dividing the middle thyroid vein and inferior thyroid artery. This procedure permitted complete exposure of the esophagus, which was lifted from the prevertebral fascia with the finger. An abscess cavity extending to the sternal notch was thus entered, containing yellow pus which on culture showed streptococcus hemolyticus and streptococcus viridans in large numbers. No gross evidence of esophageal perforation could be detected. A cigarette drain was placed at the bottom of the cavity and extended behind and somewhat to the left of the esophagus. During the next seven days the patient remained in a critical condition with continuously high fever ranging from  $38$  to  $40$  degrees C., with corresponding elevation in pulse and respirations ranging around 25. The foot of the bed was kept elevated. She was supported by infusions and by two transfusions. It was noted during this period that small portions of methylene blue given by mouth appeared on the dressings two hours later. Gastrostomy was done under local anesthesia on the sixth postoperative day. We would ordinarily have done this somewhat earlier but the critical condition of the patient as well as the minute character of the perforation caused us to delay in hopes that it might be avoided. The patient's condition steadily improved following gastrostomy, the temperature falling to normal after 48 hours. The tenderness in the neck as well as the thick yellow purulent drainage slowly decreased. On the twenty-second postoperative day the patient was allowed to sit up in a chair after being twelve days without fever. On the thirty-fourth day the gastrostomy tube was removed and mouth feedings resumed. The patient was discharged from the hospital on January 18, 1934 (thirty-eighth day). Fluoroscopic examination at this time showed no delay in the passage of the opaque mixture through the esophagus.

The X-ray studies made during the postoperative period are of considerable interest. The first film (Fig. 1), on December 17th, the day of operation, showed no changes in the lungs or mediastinum. The film (Fig. 2) taken two days later showed increased linear markings with slight, fine feathering from the fourth rib to the base of each lung, more marked on the left side. The mediastinal measurements taken at the level of the fourth posterior rib were 6.5 cms. as compared with 4.8 cm. in the first film, and were considered to indicate a mediastinal abscess. The X-ray on December 21st (Fig.



3), however, showed further increased widening of the supracardiac shadow, this time measuring 6.7 cm. The shadows in both lungs were also somewhat denser, suggesting a marked degree of chronic passive congestion. An X-ray (Fig. 4) on December 29th, the twelfth postoperative day, showed very marked clearing of the shadows in both lungs as well as decrease in the supracardiac shadow, which now measured 4.9 cms. The level of the fourth posterior rib was taken as a constant point for these measurements in the successive films.

11 NORTH GOODMAN ST.



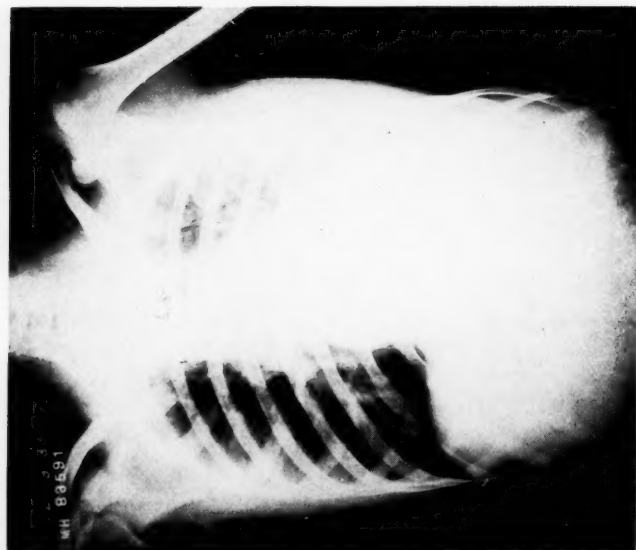


Fig. 2. Film taken two days after operation.

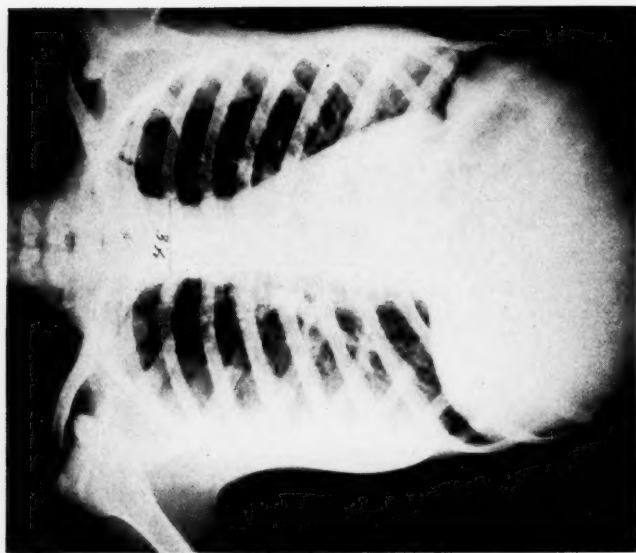


Fig. 1. First film taken on day of operation.

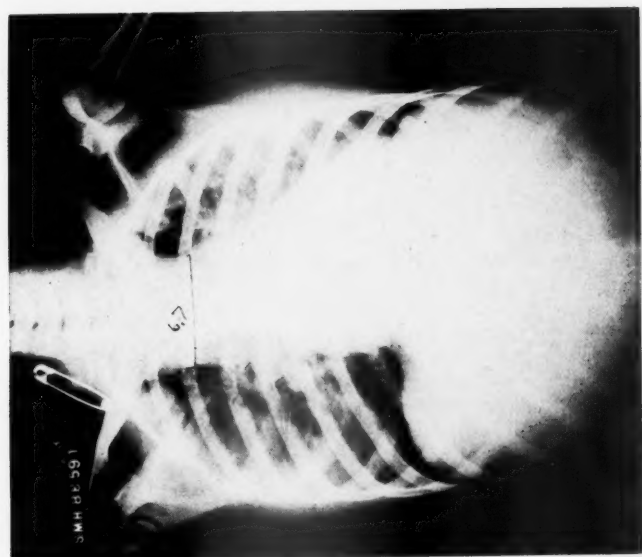


Fig. 3. Shows widening of supracardiac shadow.



Fig. 4. Shows decrease in supracardiac shadow and clearing of shadows in both lungs.

## LXXIII.

### PUTRID MEDIASTINAL ABSCESS WITH SPIROCHETAL INFECTION: REPORT OF A CASE.\*

ETHAN FLAGG BUTLER, M. D.,  
ELMIRA, N. Y.

The only object in presenting this report is to call attention to a simple procedure effectively employed in controlling a serious mediastinal infection due to typical mouth spirochetal organisms. Such infections are seen over and over again following esophageal trauma. Frequently they end fatally. This case threatened to end fatally until the alarming and apparently progressive infection was suddenly and simply brought under control.

#### REPORT OF CASE.

The patient was an adult female. During the course of an esophagoscopic examination there was trauma to the right esophageal wall at the level of the cricopharyngeus muscle. This was recognized at the time, and without delay the esophagus was exposed through a right cervical incision, parallel to the sternomastoid muscle. A small tear was found in the wall of the esophagus, and an effort was made by packing to protect the patient from infection. Mouth feeding was immediately discontinued and a few days later gastrostomy was performed for feeding purposes. Routine irrigation with Dakin's solution was instituted forty-eight hours after the operative exposure of the traumatized area. Even so, mediastinal infection was apparent on the seventh day. The exudate was the typical thin, foul, grayish-black pus, characteristic of the presence of mouth anaerobic organisms. Large spirochetes were found in abundance. By means of a lighted speculum introduced into the depths of the wound the pus could be traced to a pocket about 10 cm. deep, lying in the right posterior mediastinum. The foot of the bed was elevated and pillows removed, to facilitate free drainage. Neoarsphenamin was given intravenously in small doses. Roentgen examination (Fig. 1) indicated that the process was definitely limited. Nevertheless, the intensity of the infection increased. The patient was markedly toxic. The mouth temperature slowly rose to  $100.8^{\circ}$ . The prognosis was admittedly grave. Two days after the evidence of spirochetal infection, a soft catheter was introduced through the cervical wound, under visual control, into the mediastinal cavity and the pocket was gently irrigated with a weak aqueous solution of neoarsphenamin (0.15 grams dissolved in 50 cc. of sterile distilled water). After a few moments of contact the cavity was flushed with physiologic sodium chloride solution. The effect was dramatic in the extreme. Within twelve hours the thin, stinking, grayish

\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

From the Chest Clinic, the Arnot-Ogden Memorial Hospital, Elmira, N. Y.

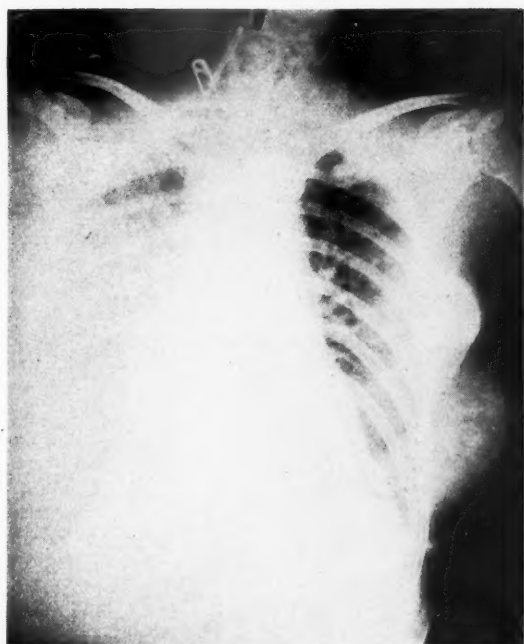


Fig. 1. Roentgen ray film of chest May 6, 1933, showing area occupied by mediastinal abscess and drainage catheters in place. The right pleural effusion is also evident.

pus had disappeared and was replaced by thick, yellow, odorless pus. Irrigation with Dakin's solution was resumed. Three or four days later considerable slough separated spontaneously from the walls of the mediastinal pocket, and the pocket itself began rapidly to diminish in size. The general condition of the patient showed marked improvement. There had been a right pleural effusion; this began to dry up. Within three weeks the esophageal tear had healed. Lipiodol mapping of the cavity then showed only a small residual sinus (Fig. 2). Within another week soft diet was being taken by mouth and the entry to the mediastinal pocket could no longer be seen. Full recovery ensued. The patient was observed at frequent intervals for the next six months and remained in all respects well.

Arsphenamin and neoarsphenamin have been administered intravenously for many years in nonsyphilitic spirochetel infections. Solutions of the same drugs in glycerin have been employed as local applications in infected mouths. Up to the present time, however, there are no recorded instances in which solutions of these drugs have been used to irrigate abscess cavities, other than those occurring in the mouth or ears.

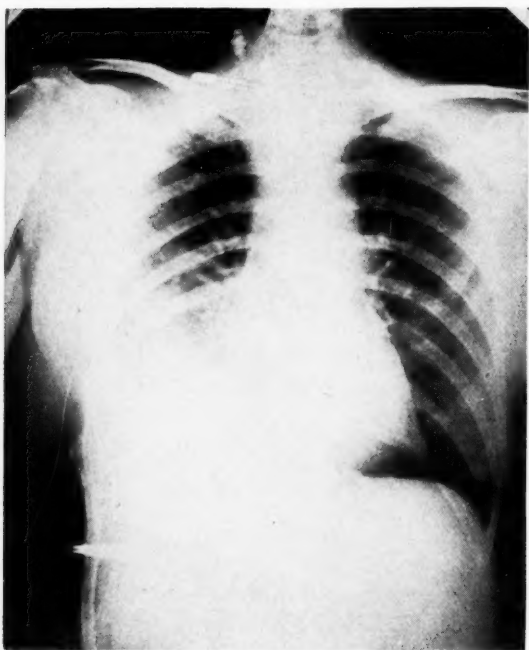


Fig. 2. Roentgen ray film of chest May 19, 1933, showing subsidence of inflammatory process, the remaining mediastinal abscess filled with iodized oil 40 per cent and the nearly complete disappearance of right pleural effusion. The right diaphragm is definitely elevated.

To draw sweeping conclusions from one case would be a serious mistake. The matter should be investigated in a well equipped experimental laboratory. On the other hand, the remarkable benefit obtained in the present case justifies this report. It may lead to more effective therapeutic measures in the management of other similar infections, whether they be pulmonary, mediastinal or located in other parts of the body.

370 WEST CHURCH ST.

LXXIV.

MULTIPLE FIBROLIPOMATA OF THE HYPOPHARYNX  
AND ESOPHAGUS.\*

RUDOLPH KRAMER, M. D.,  
NEW YORK.

REPORT OF A CASE.

A male, age 57 years, seen for the first time with Dr. S. Yankauer on December 1, 1930, complained for three months of a tickling sensation in the throat on swallowing. He had, in addition, the sensation of something being present in his throat.

The tonsils were small, half buried, and the crypts were prominent. On the posterior wall of the right arytenoid area was a spherical swelling about 1 centimeter in diameter, covered by normal mucosa, freely movable. Dr. Yankauer did a direct laryngoscopy, seized the growth in a forceps and incised it with a straight knife. The growth appeared to collapse after incision.

After the reaction had subsided, in one week, the growth was found to be smaller, but after three weeks it was about the same size as when first seen. On February 11, 1931, suspension laryngoscopy was done under general anesthesia by Dr. Yankauer. The mucosa over the tumor mass was incised and the growth exposed. It was adherent to the mucosa in the right pyriform fossa. The adhesions were separated with scissors and the growth removed. There was slight oozing but no reaction. The specimen was reported to be fibrolipoma.

Six months later I saw him again. He had had his tonsils removed by electro-coagulation in the meantime and was complaining of a sensation of a lump in the throat. This sensation was still present one and one-half years later, in April, 1933. In addition, he stated that for several weeks he had been bringing up on gagging a large mass which he described as a cold oyster. When he gagged for purposes of demonstration, a pinkish-red, flabby, club-shaped mass appeared in the mouth reaching almost to the anterior alveolar margin. The growth seemed to come from the left side of the esophageal introitus.

I did an esophagoscopy under local anesthesia. Michel clips on a long string were attached to the free end of the extruded growth, a snare was placed around the growth and pushed down to the base. An esophagoscope was passed alongside the growth, which was found attached to the posterior part of the left side of the cricoid cartilage. The growth was removed with the snare. Redundant mucosa about one centimeter square was removed from the site of the pedicle of the growth. The growth was four and one-half inches long, and was covered by normal mucosa with large veins underneath the surface. It had the typical appearance and feeling of a lipoma. Microscopic examination showed fibrolipoma. There was a slight reaction for two days as evidenced by some pain on swallowing and edema of the left arytenoid region.

\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

One week later esophagoscopy showed thickening of the mucosa of the posterior surface of the cricoid cartilage and a ridge about one inch in length at the junction of the right and anterior walls of the upper end of the esophagus.

Two months later the thickening on the posterior cricoid surface had disappeared. The ridge in the upper esophagus was now two inches in length, had a translucent surface, with a yellowish substratum and appeared to be a sessile lipoma.

This patient had first a sessile fibrolipoma on the posterior surface of the right arytenoid cartilage and the right pyriform fossa. The only symptoms were a sensation of a lump in the throat and tickling on swallowing. Secondly, two years later he had a pedunculated 4½ inch long fibrolipoma arising from the posterior surface of the left side of the cricoid. The symptoms were now a lump in the throat and an extrusion of an oyster-like mass. Third, a sessile ridge on the right side of the upper third of the esophagus, two inches in length, apparently a lipoma. In as much as there have been no

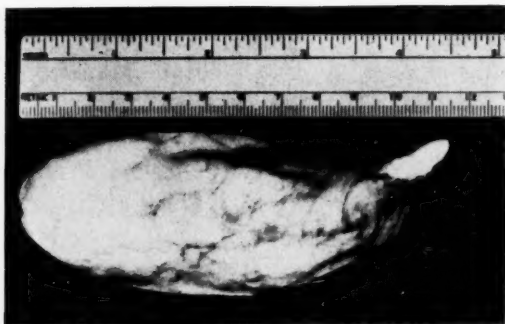


Fig. 1. Pedunculated fibrolipoma from posterior surface left side of cricoid showing dilated vessels and narrow pedicle from which mucosa stripped off on removal of growth.

symptoms from this growth, nothing has been done about it. It is possible that it may become pedunculated if it grows larger, and then removal will be much simpler than excision of a broadly attached esophageal growth.

Investigation of the literature reveals thirty-six cases of lipoma of the hypopharynx or esophagus. Six of these involved the esophagus, twenty-six the hypopharynx and in four the site of origin was doubtful—that is, from the description they seemed to be hypopharyngeal but they were reported as esophageal. Generally the patients complained of a lump or pressure in the throat and occasionally there was mentioned in addition the extrusion of a mass into the mouth

for a distance of several centimeters. In two cases marked dysphagia was present. In some marked dyspnea and even asphyxia with death resulted from the impaction of the growth in the larynx, although the patient had had no symptoms before the catastrophe. A number were found only postmortem.

Most of the cases that were discovered in life were treated endorally. A few were operated upon externally.

These tumors are interesting: (1) Because of the symptomatology which may manifest itself either by sudden death from asphyxia or by practically complete absence of complaints except those of irritation of the throat, which are often considered neurotic; (2) the bizarre, almost weird, appearance of an oyster-like or fleshy mass appearing in the fore part of the mouth and suddenly disappearing, leaving no trace of its presence.

121 EAST 60TH ST.



CONGENITAL CYST OF THE ESOPHAGUS: REPORT  
OF CASE.\*ELLEN J. PATTERSON, M. D.,  
PITTSBURGH.

While congenital cyst of the esophagus is mentioned occasionally in the literature as having been found by anatomopathologists, there are very few concrete cases reported.

Kenneth A. Phelps (1930),<sup>1</sup> speaking of congenital anomalies of the esophagus, states that "cysts of the esophagus occur as congenital anomalies but are exceedingly rare. Bittenweiser<sup>2</sup> reports such a case. There was a cyst on the anterior and right wall of the esophagus, the size of a pigeon's egg, found at autopsy on a 7-day-old child, but no other esophageal anomaly was found.

In Abt's System of Pediatrics,<sup>3</sup> Heuman reports that of fifteen cases collected by Bittenweiser thirteen were without symptoms and found at autopsy.

Birnbaum<sup>1</sup> states: "If the fistulous opening separating the esophagus and the trachea is very small or is covered by a fold of mucous membrane, then the affected persons may present no symptoms during life and the condition may be discovered accidentally at autopsy. If such favorable conditions are not present, the children die; in the cases quoted by Krause this occurred from the fifth to the seventh week. The cysts (about the size of a walnut, lined with ciliated epithelium and with mucoid contents) found in the region between the bifurcation of the trachea and the esophagus, are regarded by Krause as the degenerated remains of the communication between the trachea and the esophagus after a closure of it at both ends has taken place."

The following case has come under my observation:

## REPORT OF CASE.

CASE 1.—M. W., aged 7 weeks, had a history of inspiratory crow which the mother noticed one week after birth, with loss of weight from  $7\frac{3}{4}$  pounds at birth to 6 pounds at seven weeks of age. The babe had been breast fed for five weeks, then nursed at the breast and given the bottle with no difficulty in swallowing, and though the inspiratory crow had been present since one week after birth there was no history

\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

of cyanosis or dyspnea. The breathing was quiet at times, worse when awake and fussing. Breathing was more quiet when asleep than awake, and better when the child was held up in the arms.

The babe was well developed, though markedly undernourished with the superficial veins of the chest standing out, and some dehydration—a typical case of malnutrition.

Direct laryngoscopy, April 18, 1933, revealed the type of larynx one encounters so frequently in cases of malnutrition with the flabby, undeveloped upper aperture which improves with improvement in the nutrition, consequently I referred the child back to the pediatrician as a feeding case, with the recommendation that a roentgenogram be taken to rule out enlarged thymus.

Three days later the babe was admitted to the Children's Hospital acutely ill with bronchopneumonia, having developed dyspnea, cyanosis and vomiting of nourishment, with a temperature of  $105^{\circ}$ . A roentgenogram showed an involvement of the right lung; no enlargement of the thymus. Kahn was negative. She died April 24, 1933, aged 7 weeks 3 days.



Fig. 1. Congenital cyst of esophagus. (a) Larynx and trachea, (b) esophagus, (c) cyst, (d) lungs.

Dr. Maud L. Menten, who performed the autopsy, reported that she found a cyst that measured about 2 cm. in length and 2.5 mm. in its widest diameter, lying with the main part of the cyst to the right of the trachea, and about one-quarter lying between the trachea and the esophagus. The cyst had a clear, pearly white color and was much distended. Probes were readily passed through both the trachea and esophagus and did not enter the cyst.

When dissected out, it was found that this cyst (Fig. 1) was firmly attached to the wall of the esophagus, but there was no com-

munication between them and it had no connection with the esophagus. The laryngeal stenosis, apparently, was caused by pressure. On opening the cyst it was found to have a thick fibrous wall and to contain mucoid fluid which looked like thin boiled starch. There was nothing else in the autopsy relevant to this report except that the thymus was not enlarged, and bronchopneumonia was due to aspirated food.

The microscopic report on a section of the cyst showed a thin squamous epithelial lining with a considerable amount of striated muscle and situated deeply some fatty tissue in which were small strands of connective tissue containing nerves. The mucous glands were embedded in the muscle. The lumina of these were increased in size and the glands were larger than those found normally in the esophagus. This was probably due to pressure from the fluid contents of the cyst. The presence of striated muscle indicated that the tissue of the cyst was derived from the upper third of the esophagus, and that the esophageal glands were dilated because of pressure. The cyst probably was derived from a diverticulum of the esophagus which subsequently became closed.

The cyst must have increased very rapidly in size in the last three days of life, as there was neither dyspnea nor dysphagia when I saw the baby, three days before admission to the hospital, and direct laryngoscopy showed no evidence of pressure on the larynx or trachea.

The question of diagnosis is a pertinent one, because without symptoms of pressure, diagnosis cannot be made, and after symptoms of obstruction develop the child would probably succumb to aspiration pneumonia, as in this case. Tracheotomy in this case did not relieve the dyspnea.

121 UNIVERSITY PLACE.

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LXXVI.

MELANOMA OF BRONCHUS: METASTASIS SIMULATING  
BRONCHOGENIC NEOPLASM.\*

LOUIS H. CLERF, M. D.,  
PHILADELPHIA.

No report of primary melanoma of the trachea or bronchi has been observed in the literature. Metastasis to the lungs is common in cases of generalized dissemination, but these are commonly unassociated with clinical or roentgen evidences of bronchial obstruction. The occurrence of unexplained obstructive atelectasis involving an entire lung without evidences of a primary neoplasm or metastasis elsewhere would therefore be considered uncommon and worthy of report.

REPORT OF A CASE.

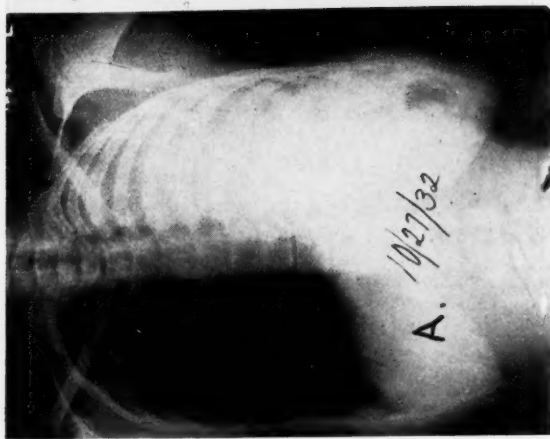
CASE 1.—Female, aged 31 years, was referred to the Bronchoscopic Clinic, Jefferson Hospital, in October, 1932, by Dr. David Kapp. She complained of unproductive cough and shortness of breath, particularly in the morning and when excited. These symptoms together with wheezing respiration began ten days following appendectomy performed under gas anesthesia in August, 1932. Because of the wheeze an asthma clinic was visited during September. All tests performed were negative, and it was believed that she was not a case of bronchial asthma. The wheeze disappeared shortly thereafter and cough remained as the most prominent symptom.

When examined by Dr. Kapp there were signs of obstruction to the left bronchus which in his judgment warranted a diagnostic bronchoscopy. On admission to the clinic the patient was comfortable. There was no wheeze. Physical examination of the chest revealed classical signs of obstructive atelectasis involving the entire left lung. This was corroborated by a roentgen study of the chest (Fig. 1A). Bronchoscopy was indicated.

At bronchoscopy the left main bronchus was found completely occluded at a point about five millimeters beyond the carina by a moderately inflamed, granular growth which appeared to be pedunculated. Following removal of a portion, it was found to spring from the posterior wall of the left main bronchus about two centimeters beyond the carina. There was considerable hemorrhage. A tentative diagnosis of carcinoma was made, although there were lacking rigidity and fixation of the bronchial wall and the tendency to bleed was more marked than is commonly observed in carcinoma. A roentgen study of the chest, made twenty-four hours after bronchoscopic removal of the growth, indicated that the left lung had re-expanded and presented a normal appearance (Fig. 1B). Physical examination corroborated these findings.

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\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.



A

Roentgenogram made before bronchoscopy. The appearances are typical of obstructive atelectasis involving the entire left lung. There was nothing observed to suggest a possible etiologic factor.



B

Roentgenogram made 24 hours after bronchoscopic removal of the growth from the left main bronchus. The obstructive phenomena had entirely disappeared and function appeared normal. (Films by Dr. J. T. Farrell, Jr.)

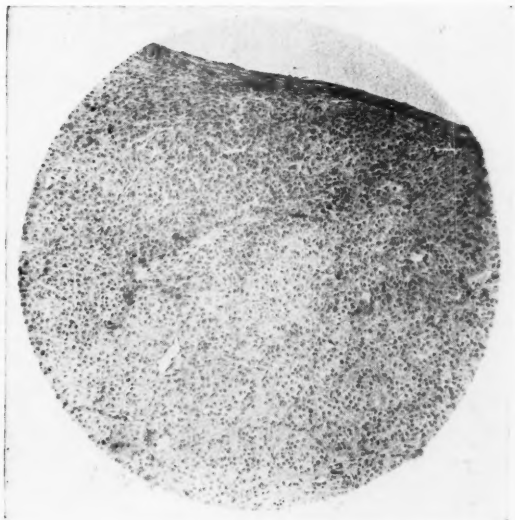


Fig. 2. Photomicrograph of tissue removed from bronchus. The growth is made up of large polyhedral cells with little stroma. There was little pigment present in the tumor tissue, but in certain areas considerable brownish pigment was observed in the tumor cells. The cells are typical of the nevus type. (Report by Dr. B. L. Crawford.)

The report of the histologic examination by Dr. B. L. Crawford was melanoma (Fig. 2). Following the receipt of this the patient was questioned regarding the presence of pigmented moles. It was learned that about one and a half years previously an irritated mole on the left arm had been fulgurated and later excised. The wound healed promptly and the incident was forgotten. A histologic section of the excised mole was secured for comparative study. Dr. Crawford found the appearances of this to be identical with those of the tissue removed from the bronchus.

A complete physical examination failed to reveal evidences of metastasis elsewhere. The site of the original growth appeared free from recurrence. The patient was kept under observation and roentgen studies were carried out at intervals. Two months later another bronchoscopy was performed. A small rounded inflammatory-like mass was found at the site of the original bronchial growth. Owing to the tendency to bleed, this was treated by electrocoagulation through the bronchoscope. Three months later wheezing respiration recurred. The roentgen findings were those of obstructive emphysema involving the left lung. At bronchoscopy the growth was found almost completely occluding the main bronchus. It was again removed with disappearance of all symptoms and roentgen evidences of obstructive emphysema. A course of deep radiation therapy was carried out. This apparently did not inhibit the rapidity of the growth. Two subcutaneous nodules appeared, one in the left infraclavicular region, the other on the left arm above the site of the original growth. The recurrence of complete bronchial obstruction necessitated bronchoscopic re-



Fig. 3. Autopsy specimen exhibiting the posterior aspects of trachea and lungs with the posterior walls of the trachea and bronchi removed. The left lung was found atelectatic. A mass of enlarged lymph nodes (A) was found between both bronchi at the angle of bifurcation of the trachea. The left main bronchus, which is obscured by the mass of lymph nodes, was occluded by new growth (B). The trachea was partially obstructed by a large nodular growth which projected from the left lateral wall (C).

movals at intervals of approximately two months. A total of eight bronchoscopies were performed. The tissue was very friable and was readily detached from the wall of the bronchus with the tip of the bronchoscope. Less hemorrhage at bronchoscopy was noted after roentgen therapy was instituted. At the last bronchoscopy performed fourteen months after the diagnosis had been established the growth had extended upward and involved the anterior and left lateral wall of the trachea. The left main bronchus was completely occluded. Although the airway was again re-established it was believed that this procedure was now hazardous. Five weeks later the patient developed a left-sided hemiplegia. Symptoms of obstruction to the airway again recurred. Bronchoscopy was not performed and the patient died during February, 1934, sixteen months after the diagnosis was made.

Autopsy findings were summarized as follows by Dr. B. L. Crawford: Metastatic melanoma of the left main bronchus and trachea with extension to the peribronchial lymph nodes (Fig. 3); metastasis to brain, ovary, lung and skin.

From the standpoint of dissemination and clinical course there seem to be two definite types of melanoma. One, in which metastasis occurs very early and is widespread, practically all organs are involved; in the other, metastasis is slow and limited and the clinical course is of much longer duration. Unfortunately there is nothing in the histologic structure of the primary lesion to indicate whether it is of a high degree of malignancy, which will terminate rapidly, or a relatively benign lesion which is of slower growth and prolonged in its clinical course. This case represents a fairly slow recurrence and metastasis of the growth with a peculiar state of dissemination. The first clinical evidence of metastasis apparently appeared in the bronchus about 1½ years after the primary growth was excised; the terminal metastases which were found at autopsy would seem to indicate that the dissemination was from the lungs and not from the original lesion in the arm. The usual type of widespread dissemination of nodules in the lung was not found in this case; instead there were only two nodules in addition to the large growth in the left bronchus and trachea.

#### COMMENT.

Apart from the surgical aspects of the treatment of moles, the remarkable distribution of the metastatic lesions and the clinical course of the disease, this case emphasizes the importance of recognizing the wheeze as a symptom of partial obstruction of a bronchus and the part that bronchoscopy must necessarily play in the diagnosis of obscure chest symptoms and signs. In addition bronchoscopy aided materially in prolonging life, although the prognosis was absolutely hopeless.

1530 LOCUST STREET.



## FIBROSARCOMA OF THE TRACHEA.\*

LYMAN G. RICHARDS, M. D., AND HARRY F. DIETRICH, M. D.,

BOSTON.

It is always with hesitation that one reports any sort of medical rarity lest it be proven later that inadequate search of literature was the only fault in failing to discover that the supposed curiosity had been previously observed on more than one occasion.

Nevertheless, I wish to record an instance of primary fibrosarcoma of the trachea in an 8 months' old infant. Tracheal tumors of all varieties are by no means common and still rarer in children. Morris Schmidt's collection of 2088 new growths of the upper air passages contained only three in the trachea. Of the 1078 malignant growths observed at the Pathological Institute of Basil there was but one in the trachea. The latest review of tumors of the trachea by Zoeller and D'Aunoy<sup>1</sup> lists a total of 351 with only twenty-six sarcomata recorded. Their own personal cases were three: one spindle cell, one round cell and one fibrosarcoma, all in patients between the ages of 20 and 53. No satisfactory etiology has been ascribable for the type of tumor beyond the possibility of its arising from misplaced muscular bands from the posterior tracheal wall (Kahler).

## REPORT OF A CASE.

Patient, a negro infant of 8 months, was admitted to the Infants' Hospital May 8, 1933, with a chief complaint of wheezing and breathlessness of one week's duration. The past history was normal.

One week ago the mother noticed the child was wheezing in her sleep. No cough, stridor, fever or vomiting. The cry had become short and hoarse. Today it refused food but did not appear particularly ill. No history of foreign body.

Physical examination showed wheezing respirations and, when the child was awakened and disturbed, asthmatic in character. The chest was resonant. Rare sonorous rales were heard. The heart was negative. The spleen was not felt. The red count was normal. The white count, 14,000. Polys 68 per cent. Tuberculin 1:1,000 negative.

After two and one-half days dyspnea continued to be marked. The child was finally transferred to the Otolaryngologic Service, where tracheotomy was done May 14th, with relief of obstruction. There was no evidence of foreign body by X-ray or story. Evidently there was some obstruction just below the larynx. Bronchoscopy was negative.

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\*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

May 25th. The temperature had gradually fallen to normal. This morning laryngoscopy and bronchoscopy were done. There were no abnormal findings. The child breathed with difficulty when the tracheotomy tube was occluded.

June 1. The child breathed easily with the tube occluded. The tube was removed, the temperature was normal, there was no difficulty. The patient was discharged June 3; the wound had healed.

July 7. The child re-entered Infants' Hospital, after discharge six weeks ago. There had been slight cough since discharge. Two days ago there were wheezing, moderate dyspnea and hoarseness. The patient was a little worse tonight and sleep was disturbed.

Respirations seemed embarrassed on both inspiration and expiration with easily audible squeaking and whistling. There was slight retraction of the lower interspaces on inspiration. The tracheotomy wound was healed. The lungs were resonant throughout. The breath sounds were harsh, well heard on both sides. Blood and urine were negative. The patient remained on the ward for two weeks. The temperature was normal the first four days, then rose to  $101^{\circ}$  for two days. The croup then gradually increased with respiratory difficulty. There was a question of obstruction due to contraction of scar tissue about the old tracheotomy wound. Following early improvement, respiratory difficulty returned and after two days became so severe as to necessitate emergency tracheotomy July 17th, with complete relief of obstruction. The temperature rose to  $104^{\circ}$  on the day of the operation. It remained high since, gradually subsiding to  $100^{\circ}$ . The respiration rate has remained between 35 and 60. Also there has developed diarrhea with from six to eight movements a day. The child was unable to breathe with the tracheotomy tube closed.

July 20. The child has run an irregular fever,  $101$  to  $102$  degrees. There are frequent soft, yellow, watery stools and some vomiting.

July 26. The patient has been afebrile for a week and has not vomited but has had some diarrhea.

July 27. Transfusion of 150 cc. was given.

August 3. Diarrhea has still persisted, as many as eight watery stools a day. The formula was changed. There was fever during past five days with high pulse and respiratory rate up to sixty. The child is dehydrated; another transfusion given.

August 10. Diarrhea continues. The temperature to  $103^{\circ}$  until four days ago, is now down to  $100^{\circ}$ . The child is taking 1,000 cc. of fluid a day. Another transfusion was given, August 9th, of 160 cc. The child breathes fairly well at times with the tube occluded. The child is losing ground, even though temperature is subsiding.

August 17. Diarrhea continues, though not so severe. There is still some fever. Weighs 13 pounds. The child seems brighter than during past few weeks. The tracheotomy tube was removed without causing any obstruction.

August 24. Normal temperature continued since the last note; the stools decreased and were of normal consistency. Gaining weight. Breathing well without tracheotomy tube. Slight bronchial rattle. Acute otitis media developed today, requiring paracentesis.

September 7. After gaining weight to 14 pounds, temperature rose to  $105^{\circ}$ , respirations to 70, pulse to 170; right ear bulging and incised. Fever the next four days from  $101$  to  $104$  degrees; respirations 17 to 40. Another transfusion given. Six days ago temperature dropped to normal, respirations to 30, and has been afebrile since.

September 14. Entirely afebrile since last note. Taking food well. Gaining. Noisy respirations. Laryngoscopy performed yesterday. No definite abnormality of larynx or upper trachea found.

September 21. During the past week patient has had marked dyspnea with inspiratory and expiratory obstruction, more difficulty in expiration than inspiration. Temperature normal until four days ago, then  $101^{\circ}$ . More diarrhea has developed. A large amount of mucus sucked from trachea. More comfortable. Weight, 15 pounds 12 ounces.

October 5. Condition unchanged. Irregular fever up to  $103^{\circ}$ . Some clouding of mastoids. Gaining weight.

October 10. Tracheotomy tube reinserted. Moderate difficulty in breathing through tube and daily discharge of a small amount of blood through tube. Frequent suction without alleviating respiratory distress. Normal temperature for the past two weeks. Respirations from 25 to 70.

November 2. Normal course until yesterday, when great difficulty in breathing, coughing up a large amount of blood through the tube. Suction obtained an ounce of bloody mucus. Respirations more easy. Same difficulty again today. Suction again applied; an ounce of blood obtained.

November 9. Still coughing up blood through the tracheotomy tube. Breathing more quiet.

November 23. Child on downhill course. During the past four days severe attacks of difficulty in breathing. Coughed up a moderate amount of red blood particles in which are granulation tissue. No weight gain. Taking food poorly. Temperature  $99$  to  $105$  degrees.

November 29. Bronchoscopy with findings of small amount of bloody mucus in trachea. Nothing abnormal except that left main bronchus seems smaller than normal. Following bronchoscopy, long No. 3 trachea tube inserted, child seemed to breathe better. In the evening of November 28th breathing quietly and without difficulty. Shortly after midnight was still apparently breathing easily. Respirations suddenly ceased.

#### REPORT OF THE AUTOPSY.

The body is that of a poorly developed, emaciated female negro infant measuring 72.5 cm. in length. Moderate pallor of the mucous membranes of the lips, gums and buccal cavity is present. In both the anterior and posterior triangles of the neck smooth, firm, freely movable lymph glands are palpable. Three centimeters above the manubrium is a tracheotomy wound which measures about 1 cm. in diameter. A zone of dense cicatrization surrounds the pale, glistening lining of the wound. No purulent material is present, but slight pressure on the trachea forces a considerable amount of frothy sanguinous fluid into the wound.

*Peritoneal Cavity.*—The only relevant abnormality noted on examining the contents of the peritoneal cavity in situ is the relative enlargement of the liver. A smooth liver edge is visible 4 cm. below the xyphoid and 5 cm. below the costal margin in the right mid-

clavicular line. The mesenteric glands are small and of normal consistency.

*Pleural Cavities.*—Both pleural cavities contain a small amount of slightly cloudy yellow fluid, in which are seen a few flakes of fibrin. The lungs do not collapse when the chest plate is removed.

*Organs of the Neck and Lungs.*—Removed en masse.

*Larynx.*—The laryngeal mucosa is slightly edematous and somewhat injected. No ulcerations are present.

*Trachea.*—Immediately below the larynx is seen the tracheotomy wound. (Fig. 1, No. 1\*). About the edges of the latter the mucosa is somewhat redundant. Just above the bifurcation of the trachea are three areas of ulceration of the mucosal surface. These are shallow, markedly irregular and partially filled with exudate and debris. No recent hemorrhage is evident. Attached to the left wall of the trachea, and at the level of the tracheotomy wound, is an irregular, roughly globular gland (Fig. 1, No. 2), which measures 1.4 cm. in diameter. Three millimeters below this, and attached to it in its deeper portion, is a large, slightly oval mass (Fig. 1, No. 3), measuring 3 x 1.5 x 1 cm. The medial portion of the latter is incorporated in the wall of the trachea. It appears to have destroyed a portion of the cartilaginous ring, and protrudes slightly and irregularly into the lumen of the trachea. Erosion of the tracheal mucosa over this mass is evident. The lower pole of the tumor is lodged in the angle formed by the trachea and the left main bronchus. Cut surfaces of this mass are pale, gray white, homogeneous and of the appearance of very dense, avascular fibrous tissue. Anteriorly, the tumor is in contact with, and connected to, a large irregular mass (Fig. 2, No. 4), which measures 3.5 x 2.0 cm., and which covers the entire anterior external surface of the lower trachea. Cut surfaces show irregular pinkish to yellowish-gray areas separated by very pale gray-white trabeculae. Adherent to the upper pole of this latter mass is a moderately soft, pinkish gray, oval gland (Fig. 2, No. 5), which measures 1 x 1.4 cm. At the bifurcation of the trachea there is an irregular, lobulated, tenaciously adherent mass of glands (Fig. 1, No. 6), measuring 2.8 x 2 x 1 cm., which extends along the right main bronchus to the hilus of the lung. Surfaces made by cutting show a mottled surface of dark reddish-gray and paler yellowish-gray. The paler areas are the more firm. At the hilus of the left lung are several discrete glands, measuring .3 to .8 mm. in diameter, which are similar to those just described.

\*The numbers refer to the indicated areas on Figs. 1 and 2.



Fig. 1. Photograph of gross specimen viewed from the posterior aspect. 1, tracheotomic wound; 2 and 3, tumor; 4, 5 and 6, enlarged and inflamed lymph nodes.

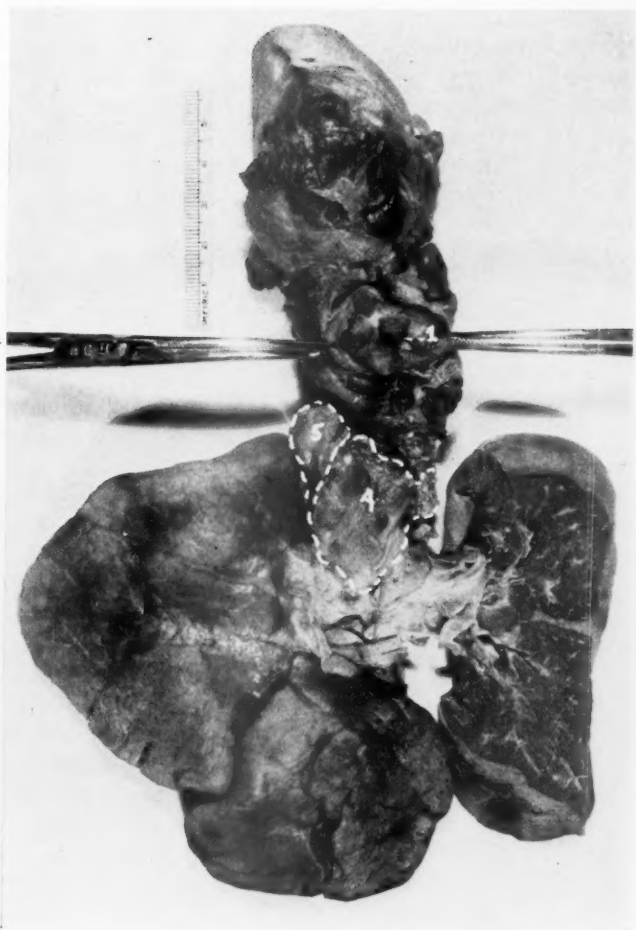


Fig. 2. Photograph of gross specimen viewed from the anterior aspect. 1, tracheotomic wound; 4 and 5, enlarged and inflamed lymph nodes.

The tumor, together with the adjacent glands, practically encircles and definitely narrows the lower tracheal lumen. Glands have similarly encroached on the lumen of the primary bronchi. The lumen of the left primary bronchus is particularly narrowed.

*Lungs.*—With the exception of the bases of the lungs, where slight fibrinous roughening is present, the pleural surfaces are smooth and glistening. Both lungs are emphysematous anteriorly; posteriorly the parenchyma is dark reddish-purple, increased in consistency and decreased in crepitaney. Alveolar markings are largely effaced. Throughout the posterior portion of the right lower lobe there are very many grayish-white rounded miliary lesions and, in addition, several firm, irregular, yellowish, discrete and confluent nodules, which measure 2 to 3 mm. in diameter, are noted. The appearance of surfaces made by cutting substantiates the impressions of emphysema and pneumonia, and reveal many of the small gray nodules deep in the parenchyma. The primary and secondary bronchi contain a large amount of frothy, tenacious, serosanguinous fluid. The mucosa is reddened and in the right primary bronchus is interrupted by several small areas of ulceration.

#### MICROSCOPIC EXAMINATION.

*Tumor.*—Sections of the main tumor (Fig. 1, No. 3) and the immediately adjacent mass (Fig. 1, No. 2), stained with hematoxylin-eosin, anilin blue and phosphotungstic acid—hematoxylin, show a relatively slowly growing fibrosarcoma which apparently arises in the tracheal wall and which shows acute and chronic inflammatory changes in that portion bordering on the tracheal lumen. The tumor has largely effaced the architecture of the tracheal wall in this area, and only a few small fragments of mucosa and rare, distorted tracheal glands are seen. (Fig. 3.) One section shows several fragmentary inclusions of smooth muscle. The tracheal surface of the tumor is abundantly infiltrated with large numbers of polymorphonuclear leucocytes, lymphocytes and plasma cells. Vascular congestion is marked.

The larger portion of all sections is occupied by slender, elongated cells with relatively large oval vesicular nuclei, scanty cytoplasm and polar strands of moderately fine fibrillæ. (Fig. 5.) The cells are roughly arranged in undulating, interdigitating bands. (Figs. 3 and 4.) Interspaced between these strands are collections of slightly less well differentiated cells; rarely mitotic figures are seen among the latter groups. The special stains accentuate a considerable amount of



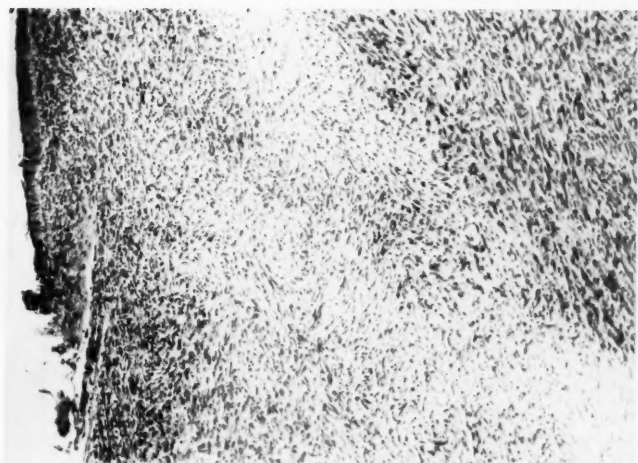


Fig. 3. Photomicrograph of the wall of the trachea. The complete effacement of the normal histology by tumor, and the ulceration in the tracheal mucosa, are to be noted. Hematoxylin and eosin stain; reduced from a magnification of X 112.

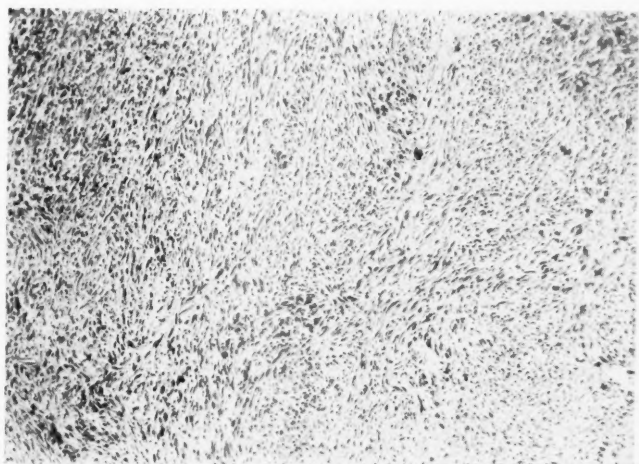


Fig. 4. Photomicrograph of tumor. Note general architecture. Hematoxylin and eosin; reduced from a magnification of X 112.



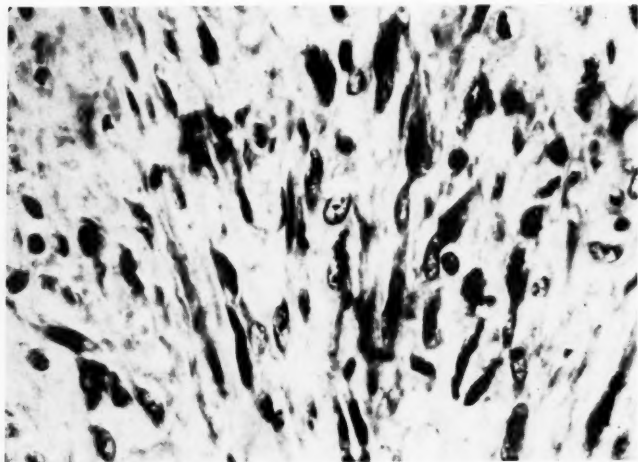


Fig. 5. Photomicrograph of tumor. Note bundles of fibrillae at either pole of the elongated cells, and the diffusely distributed intercellular substance (collagen). Aniline blue stain; reduced from a magnification of X 985.

diffusely distributed intercellular substance which has the appearance of collagen.

*Regional Lymph Glands.*—Representative sections from each of the other masses (Fig. 1, Nos. 4, 5, 6) and from the lymph glands at the bifurcation of the trachea, and the hila of the lungs, fail to show any evidence of tumor involvement. The microscopic picture in each is similar, and is characterized by those changes which are regularly associated with acute and chronic inflammation. The capsules show definite thickening and are infiltrated with moderate numbers of lymphocytes and plasma cells. Within the peripheral sinuses and throughout the parenchyma are large numbers of polymorphonuclear leucocytes and plasma cells. Lesser numbers of mononuclear leucocytes and rare eosinophiles are seen. The secondary follicles are composed of densely packed, darkly staining, relatively mature lymphocytes, and about their peripheries show considerable acute inflammatory reaction. Fibrous trabeculae are, by their breadth, unusually conspicuous, and in addition show considerable edema and diffuse infiltration with moderate numbers of polymorphonuclear leucocytes, plasma cells, eosinophiles and lymphocytes. (Fig. 6.) Vascular engorgement is prominent in all sections. In occasional fields small amounts of free and phagocytosed hemosiderin are seen.

*Retroperitoneal Lymph Glands.*—Several sections fail to show any evidence of tumor metastasis.

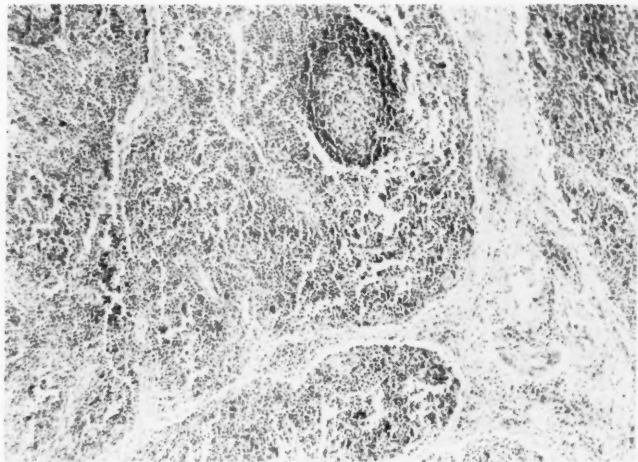


Fig. 6. Photomicrograph of regional lymph node (Fig. 2, No. 4). Note increase of fibrous tissue. There is no evidence of metastasis. Hematoxylin and eosin stain; reduced from a magnification of X 112.

*Lungs.*—Seven sections, exclusive of those stained with Scherlach R, show widespread acute and chronic pneumonia, acute aspiration pneumonia with foreign body reaction, and emphysema of the severity usually associated with partial obstruction. In many fields the pleura is markedly thickened, edematous, congested and infiltrated with polymorphonuclear leucocytes. The pathologic changes seen in the pulmonary parenchyma may be considered characteristic of three distinct processes:

1. Chronic interstitial pneumonia: Although intra-alveolar exudation is prominent, the chronic interstitial involvement is more widespread. In many fields, and particularly prominent in the peribronchial regions, the alveolar walls are thickened by an increase in fibrous tissue and a diffuse infiltration with moderate numbers of mononuclear leucocytes and lymphocytes and occasional plasma cells. Eosinophiles are relatively rare.

2. Acute bronchopneumonia: In large irregular areas, the increased bulk of the alveolar walls is occasioned by vascular congestion, edema and infiltration with polymorphonuclear and mononuclear leucocytes. The character of the alveolar exudate presents those variations associated with an acute bronchopneumonia. In some fields it is largely composed of mononuclear leucocytes; elsewhere the alveoli are filled with polymorphonuclear leucocytes, red blood cells, precipitated serum, or mixture of all of these.

3. Acute aspiration pneumonia: In well circumscribed areas the alveoli contain vacuolated phagocytic large mononuclear cells. Interspersed between these cells, as seen in the Scharlach R preparations, are large and small, unphagocyted, deep red and orange fat droplets. In many of these fields the cells of the interstitial infiltration contain phagocyted fat droplets. In H. and E. sections, many highly refractile, light yellow globules, which apparently have excited no inflammatory response, are seen. Rarely certain of the fat droplets have caused a definite foreign body reaction and, in addition to the large mononuclear phagocytes, multinucleated giant cells are seen. The findings are consistent with the aspiration of milk, a second more irritating organic fat, and a nonirritating inorganic oil.<sup>2</sup> Throughout all sections are numerous air bubbles entrapped in the alveolar exudates and occasionally a narrow band of hyalin-appearing exudate is closely applied to the alveolar wall in the form of an asphyxial membrane.<sup>3</sup> In areas which are free from exudate the alveoli are markedly distended and large, irregular, emphysematous spaces are formed by the distortion and rupture of intervening alveolar walls. The bronchi contain varying amounts of the various types of exudate and all sections show the bronchial walls infiltrated with large numbers of polymorphonuclear leucocytes. About many of the bronchi are small areas of erythropoiesis. The interlobar fibrous trabeculae are largely free from cellular infiltration but are markedly edematous. Many small venules occluded by septic thrombi are seen; rarely small lymphatics distended with polymorphonuclear leucocytes are also observed.

*Spleen.*—Two sections show marked congestion and slight acute splenitis.

*Liver.*—Several sections show moderate congestion and slight fat infiltration. Throughout most of the fields the histology of the liver cells and the orientation of the liver cords are unaltered; in sharply delimited central areas, however, there are fatty changes in the cells. Two fields show minute foci of necrosis.

*Final Diagnosis.*—Tracheotomy wound.

Primary fibrosarcoma arising in wall of trachea.

Ulcerations of the trachea.

Ulcerations of the esophagus.

Chronic bronchopneumonia—bilateral.

Acute bronchopneumonia—bilateral.

Aspiration pneumonia—right middle and lower lobes.

Acute fibrinous pleuritis.

Acute toxic splenitis.

Slight fatty infiltration of the liver.

Acute accidental involution of the thymus.

Atheromatous changes in the aortic leaflet of the mitral valve and malfixation of the ascending colon.

COMMENT.

In retrospect the history is that of repeated and variable tracheal obstruction which was apparently due to extrinsic factors. Non-localizing signs and symptoms referable to an acute inflammatory process several times confused the picture. During the six and one-half months prior to its death, this infant had repeated attacks of tracheal obstruction which were entirely relieved by tracheotomy. Bronchoscopy and roentgenography revealed no apparent cause for the symptoms. Several times high fever and either elevated or markedly reduced white blood cell counts were present without demonstrable cause.

The origin in the trachea of a tumor of the type described must be extremely rare. Neither Schultz<sup>1</sup> nor Ewing<sup>2</sup> in their discussions of fibrosarcoma mention an instance of it. Because of its relatively slow rate of growth and the absence of metastases, the occurrence of a similar tumor in a more accessible locus would presumably permit successful operative interference. Its origin in the wall of the trachea and the expansion outwards prior to erosion into the lumen, precluded an early diagnosis. Had not secondary infection intervened it is probable that the ultimate expansion of the tumor would have made its presence recognizable, but when its dimensions had assumed sufficient proportions to permit diagnosis operative treatment would no doubt have been unsuccessful.

The sequence of events in the case under discussion may, we feel, be deduced. At some time during the early stages of the growth of the tumor the child had a coincidental attack of diffuse bronchopneumonia; residual infection provoked the hylus and mediastinal lymphadenopathy. It has been shown by Farber and Wilson<sup>3</sup> in this clinic that chronic pneumonia causes enlargement of the regional glands, and that after some time, with or without several acute exacerbations of the pneumonic process, the glands may cause a definite partial obstruction to one or several secondary bronchi. Depending on the degree and type of obstruction so instituted, emphysema or partial atelectasis may result; always at this stage there is evident a predisposition to frequent exacerbations of a more or less acute nature.

As the glands at, and particularly above, the bifurcation of the trachea responded to chronic infection by permanent enlargement and then further temporary increases in size, due to acute inflammatory reaction occurred, obstruction reached a degree sufficient to cause temporary respiratory embarrassment. That this obstruction was effective in the lower trachea and that it was there caused by the coincidental occurrence of the tumor and the glands, is attested to by the prompt and complete relief of the symptoms by tracheotomy. Partial obstruction of the bronchi by glands alone is suggested by a study of the pathologic specimen. When, because of enfeeblement due to repeated infection, the child began to aspirate food and vomitus, a more or less terminal picture of aspiration pneumonia was superimposed on the existing pathology. Review of the history indicated several episodes of aspiration and in addition disclosed that both cod liver oil and mineral oil had been given. The largest amount of fat seen in the sections was, of course, accounted for by the aspiration of milk.

#### SUMMARY.

These findings were a profound surprise to all those who had been associated with this prolonged and trying case. The definiteness plus the rarity of the lesion may have caused the pathologist some satisfaction, but it did little to explain the clinical signs as observed by the bronchoscopist. The latter derived some slight satisfaction in the fact that the tumor mass did not to any appreciable extent encroach on the lumen of the trachea and hence was not visible to bronchoscopic examination. What should perhaps have been detected was the mucosal ulceration of the tracheal wall, from which in all probability the bleeding originated. This ulceration was adjacent to the main tumor mass, was undoubtedly caused by it rather than by any pressure from the lower end of the tracheal cannula.

What still remains without satisfactory explanation is the three distinct attacks of laryngeal or subglottic obstruction, each relieved by tracheotomy, and two of which resolved spontaneously to such an extent as to permit ready decannulation. Intermittent swelling of the encircling masses may have been the etiologic factor, but direct external pressure on the tracheal lumen to the extent of persistently encroaching on this was not manifest at the autopsy.

The appearance and position of the tumor masses are obviously such as to raise the question whether they were not after all lymph nodules in this region, which, becoming involved in a malignant growth, finally brought the case to a fatal termination.

It must be noted, however, that the tumor is not lymphosarcoma and that no other lymph nodes in the body were similarly involved, as would have been the case in an instance of true lymphosarcomatosis. Moreover, there are sections from other near by lymph nodes which show only chronic inflammation. The main tumor growth is here an intimate part of the tracheal wall, directly beneath the ulcerative mucosal surface. Rare as this finding is, it is the opinion of expert pathologists that we are dealing with a true fibrosarcoma of the trachea.

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ABSTRACTED SCIENTIFIC PROCEEDINGS OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

Fortieth Annual Meeting, Charleston, S. C., April 3-5, 1934.

SYMPOSIUM ON NEWER CLINICAL APPROACHES IN OTOLARYNGOLOGY.

1. NUTRITIONAL INFLUENCES. William Weston, Columbia, S. C.

The effects of mass dietary factors as observed in the Japanese people were discussed. It is probable that the Japanese come nearer to adhering to what we understand is a satisfactory diet than any other people in the world. As a result, they exhibit such characteristics as mental alertness, great energy, remarkable physical strength and extraordinary endurance.

The author reviews our knowledge of the effects of diet upon the mucous membrane and the special senses. He emphasizes the relation of certain mineral elements upon the utilization of other mineral elements by the body. In conclusion, the point is made that it is neither necessary nor advisable to resort to artificial sources for the materials from which to select our diets.

2. SYMPATHETIC INFLUENCES (AUTONOMIC LEVEL). Francis B. Blackmore, M. D., Columbus, Ga.

There are two antagonistic parts of the autonomic nervous system. One may visualize extreme sympathetic dominance at a high level and extreme parasympathetic dominance at a low level. Then the condition of the patient at a given time can be indicated as being at some level between these two. Such a level is named the autonomic level by the author.

Symptoms and signs characteristic of each type of displacement are recognized. Numerous factors exerting displacing force in each direction have been observed. When properly selected and applied to the patient, these factors will exert force toward the establishment of a normal equilibrium.

3. INSULIN THERAPY. Samuel M. Beale, M. D., Sandwich, Mass.

The author discusses a series of clinical observations of the action of insulin beginning with the improved growth of a toe nail in a dia-



betic. This suggested its usefulness in sclerotic diseases—cell degeneration. Further observations were made in cell perversions—benign and malignant growths, and in defects of calcium metabolism. The application of all the above was made to otology, rhinology and laryngology.

#### DISCUSSION.

DR. D. C. JARVIS, Barre, Vermont: The chemical content of the individual's food is of primary interest. An excess of potassium and iodine in organic form produces a watery nasal discharge. With too much sodium chloride a dry nose develops.

In regard to Dr. Blackmore's paper, we have been more or less familiar with facts relating to the autonomic nervous system, but the difficulty has been in applying these facts clinically. When the sympathetic is dominant, iodine in some form seems to be the therapeutic agent of choice, and when the parasympathetic is dominant dilute hydrochloric acid is indicated.

Ulceration of tissue, mucopurulent discharge, scab formation within the nose and new growths are all seen in the light of a low tolerance to carbohydrates. In some cases, the carbohydrate intake is kept low during surgical convalescence, in others powdered spinach is also added, while in a third group showing dried nasal secretions, four units of insulin daily is advised.

DR. IRA FRANK, Chicago: Dr. Weston's paper supplies much food for thought. I am greatly impressed by the large number of poisonous fungi which may be found in cereal grains. A deficiency of the vitamins causing various conditions has been stressed. As Dr. Weston suggests, an insufficient supply of vitamin D may cause a malformation of the malleus, incus and stapes.

Dr. Blackmore's paper is particularly interesting because it deals with the pale septum which I have previously discussed as possibly affording some hint of faulty body chemistry. I have found the pale septum to have suggestive value in diagnosis and in treatment, but I realize that a vast amount of work remains to be done on the subject.

DR. T. W. MOORE, Huntington, W. Va.: Dr. Weston has shown how the mineral and vitamin properties of milk may vary greatly. His paper shatters many of my ideals in a most convincing manner.

Dr. Beale's paper astounds us with the number of varied diseases in which he has found insulin useful. I agree with Dr. Beale's suggestion that primitive peoples are free from cancer and that this is probably due to the kind of food eaten and its method of preparation. Will Dr. Beale tell us if he has any guide in the administration of insulin? Has he not had cases developing hyperinsulism?

DR. JOHN F. BARNHILL, Miami Beach, Fla.: Dr. Weston this morning gave very beautifully the story of the Canadian refugees from the United States, those who went to Canada and those who went to the Bahama Islands or to the Caribbean Sea islands. He said that the health of the Canadians was so much better and their mentality so much better than that of those in the Caribbean Sea islands. But their environment was not taken into account. In the Caribbean islands their environment was 95 per cent Africans and their mentality was not likely to develop as it would among the Canadians. Further, in the Caribbean islands there is very little of what is known as modern sanitation. Also, these people eat only fish, bananas and grapefruit.

DR. H. M. TAYLOR, Jacksonville, Fla.: A patient was relieved by the regime he was placed on for sneezing, postnasal dripping, bad breath, etc., and whose nasal



membrane was very red. Alkalis, spintrate and green vegetables were given in abundance. Carbohydrates and meat were cut down.

DR. WILLIAM WESTON (closing): I have been particularly interested in the iodine element of our food because a study of this subject has revealed the fact that iodine was very important in the diet of all people who achieved greatness, or in nations that survived. It is important to know that it is only from the organic form of any element that one receives its ultimate benefit. The food must contain a sufficient amount of amino-acid tyrosine in order that inorganic iodine may be as effective as its organic form. If the diet contains a sufficient amount of iodine one can get a perfect balance in calcium, phosphorus and magnesium, and in no other way.

DR. FRANCIS B. BLACKMORE (closing): I wish to emphasize that we have not paid sufficient attention to the observations of the old clinicians. I would advise reading Dr. Cannon's book on "Bodily Changes in Pain, Hunger, Fear and Rage."

DR. SAMUEL M. BEALE (closing): The question was raised as to whether there are deleterious effects from persistent use of insulin in these nondiabetics, and I shall have to say, Yes. The dose is small, however, and the dangers are small in proportion. The primary effect is on the endocrines and the effect on the blood sugar is secondary.

#### 4. ACCIDENTAL INJURY OF THE LATERAL (TRANSVERSE) SINUS DURING MASTOIDECTOMY. Henry Dintenfuss, M. D., Philadelphia, Pa.

An effort has been made to study and correlate data concerning accidental injuries of the lateral sinus during mastoidectomy. Injury of the lateral sinus increases the risk of the mastoid operation.

When the mastoid is large and pneumatic in type the space between the lateral sinus and the posterior canal wall is broad, injuries of the sinus are less apt to occur, therefore, than in the small sclerotic mastoid where the space between these structures is narrow. Sinus injuries appear to be more frequent in the right mastoid in men and in the left mastoid in women. Absence of the sinus plate or adhesions of the sinus to corresponding structures predispose to sinus injury.

The employment of the curette in bone exenteration is more likely to be followed by trauma to the lateral sinus than that of any other instrument. The use of the gouge is attended with least danger. It would seem wise to avoid the use of the curette, especially the narrower ones, when exenterating bone in close proximity to the sinus.

Bone in the immediate sinus area should be removed last in all mastoid operations, for if the sinus is traumatized the danger of infection is less, all the diseased tissue with infecting bacteria having already been removed. There is also the advantage that the operative procedure will not be interrupted by hemorrhage.

The complications of sinus injury are principally hemorrhage, secondary sinus rupture, septic thrombophlebitis and air embolism. Infection following injury of the sinus is usually due to the virulence of the organism of the diseased tissues. Infrequent change of packing is another contributing factor. Thrombi in the lateral sinus following accidental injury may become organized and so remain, or they may disappear spontaneously by secondary absorption, the sinus regaining its patency.

The treatment of sinus injury consists primarily of arresting hemorrhage by tamponing the mastoid cavity, then of obliterating the sinus lumen by introducing gauze between the sinus and its bony covering above and below the site of injury. Blood stream infections should be controlled by intravenous medication, blood transfusions and jugular ligation.

It is probable that the occurrence of septic thrombophlebitis following mastoidectomy is a sequence, in some instances, to accidental injury of the sinus not recognized at operation.

#### DISCUSSION.

DR. DAVID N. HUSIK, Philadelphia, Pa.: I am heartily in accord with all Dr. Dintenfass has said. I do not think the linea temporalis is a good surgical landmark for the location of the lateral sinus. X-ray plates do, however, tell us the position of the sinus. I do not uncover the sinus unless I am suspicious of a thrombus.

DR. HORACE WILLIAMS, Philadelphia, Pa.: One wonders that the lateral sinus is not injured more often. My opinion has always been that there are no ill effects from removal of the sinus plate, provided there is no injury to the sinus.

DR. C. McDOUGALL, Atlanta, Ga.: I have learned not to uncover great areas of the sinus. I think the more the sinus is uncovered the greater the chance for infection. I have had the misfortune to puncture two sinuses. One healed over without complications; in the other case the patient finally died and at postmortem a clot was found in the longitudinal sinus.

DR. HENRY DINTENFASS (closing): In the majority of cases the oblique position of the linea temporalis means a narrow mastoid, and a narrow mastoid always indicates a forward position of the lateral sinus. I have come across several instances in which the lumen of the sinus has been restored following thrombosis of the vessels, and this has been proven by autopsy.

#### 5. LARYNGOPULMONARY TUBERCULOSIS: A REVIEW BASED ON TWENTY YEARS' EXPERIENCE. J. P. Dworetzsky, M. D., Liberty, N. Y.

Tuberculosis of the larynx should be considered as a complication of pulmonary tuberculosis and should never be viewed nor treated as a clinical entity. The treatment of laryngopulmonary tuberculosis should consist mainly of the treatment of the parent

lesion, and that is the pulmonary disease. Collapse therapy, which is the most valuable measure in the treatment of pulmonary tuberculosis, when successful, will not only prevent the development of laryngeal tuberculosis but will also promote healing of a co-existing laryngeal lesion.

A group of 500 cases of pulmonary tuberculosis was examined and compared with a fairly similar group of 500 cases examined twenty years ago. Whereas the incidence of laryngeal tuberculosis in 1914 was 25.6 per cent, the incidence in 1934 was found to be 14.6 per cent. This pronounced reduction of the occurrence of laryngeal tuberculosis can readily be explained by the improved methods of diagnosis and the consequent earlier application of treatment; also by the improved methods of treatment. Whereas collapse therapy was not employed in any of these cases in 1914, it was employed in 20 per cent of the cases of the present group.

#### DISCUSSION.

DR. THOMAS E. CARMODY, Denver, Colo.: I do not believe that we ever see cases of primary tuberculosis of the larynx. The cases that have been reported as such have not been thoroughly examined. As a rule, in cases of progressive tuberculosis of the larynx, we find progressive pulmonary tuberculosis. In the diagnosis of tuberculosis of the larynx there is no question that we may have other diseases of the larynx also present, such as cancer, syphilis, etc.

The fact that pulmonary lesions are treated better today than they were twenty years ago is the reason why laryngeal disease is less frequent. In the local treatment, sprays are of very little value except for cleansing. Artificial pneumothorax is of great value; however, sunlight and artificial sunlight have been overdone. Cautey does a great deal of good, but for the removal of the epiglottis and surgery of that kind, we rarely use it today.

DR. E. A. LOOPER, Baltimore, Md.: At the State Sanatorium we have discontinued the use of all chemical caustics and have found actual cauterization most satisfactory, when used on properly selected cases and if general treatment of the pulmonary lesion is actively carried out.

It is unfortunate that such a pessimistic general opinion still persists, even at this late date, in regard to the treatment of laryngeal tuberculosis. If recognized early, most of the lesions may be cured. Of 3,227 patients at the Maryland State Sanatorium in 1928, we found 15.5 per cent developed laryngeal lesions. By the use of the cautey we have been able to improve or cure 65.5 per cent of the cases who had moderate lung involvement and 26.5 per cent with far advanced lung involvement were improved.

DR. PERRY G. GOLDSMITH, Toronto, Canada: I think there is a great deal of looseness in the diagnosis of tuberculosis of the larynx. A person who has tuberculosis of the larynx always has a change in the mucous membrane of the pharynx; a redness and inflammation; and the same condition exists in the trachea. To diagnose tuberculosis of the larynx there must be infiltration and ulceration.

I am not quite prepared to accept the statement that collapse therapy is a prevention of laryngeal tuberculosis.

As to treatment, if a patient has progressive tuberculosis, copious sputum and diffuse lesions of the larynx, let the larynx alone. A tracheotomy is a godsend in advanced cases with edema. Local treatments have long been given up except in the form in which the cautery is used. Cautery is not advisable in progressive laryngeal tuberculosis. Such a patient will die of his pulmonary condition or his general condition. Vocal rest in a laryngeal lesion is very essential. I have not had much experience in heliotherapy. I do not use it.

DR. JULIUS P. DWORETZKY (closing): I do not think there is much doubt that collapse therapy prevents laryngeal tuberculosis. Occasionally we have to do a tracheotomy, but we have a great deal of trouble with these cases.

6. A STUDY OF AURAL TUBERCULOSIS (Candidate's Thesis). John Miller, M. D., Greenwich, Conn.

A total of 974 patients with pulmonary tuberculosis were examined, and of this number 102 had chronic otitis media. An incidence of tuberculous ear infection of 1.7 per cent was found among patients with pulmonary tuberculosis. Nearly all had positive sputa. Three per cent of tuberculous adults have simple chronic purulent otitis media. The greatest number of tuberculous otitis cases were among young adults. No multiple perforations of the membrana tympani were seen. The infection of the middle ear is direct from active pulmonary lesions by way of the nasopharynx. A painless, very deaf, scantily discharging ear in a young tuberculous adult is suggestive of tuberculous involvement. The ailment is relatively benign in adults, but loss of auditory function is pronounced and permanent.

In no case under observation was any surgical interference considered feasible or necessary. No vestibular disturbance was noted. Diagnosis cannot be arrived at clinically, the most reliable methods being animal inoculation of suspected material or microscopic examination of granulations removed from the ear or mastoid.

DISCUSSION.

DR. JAMES G. DWYER, New York: My interest has been from the pathological standpoint, in trying to develop a method of isolating the organism directly by culture. Examination of tissue from the ear is very unsatisfactory. Furthermore, I do not think it is a very good policy to cut the tissue out for examination. We have carried out the method of planting the culture by direct isolation, according to Petroff's method.

I think most of the infections are blood-borne or through the lymphatics, and not through the eustachian tube. In children the ear infection is not secondary to a lung lesion and is usually the bovine form. In 1916 or 1917, St. Clair Thomson made the statement that he had never seen caries of the ear, that he had never seen ossicles coming out, or facial paralysis or multiple perforation. Our experience is directly opposite to that.

DR. J. P. DWORETZKY, Liberty, N. Y.: My experience with tuberculosis of the middle ear has been entirely limited to adult patients. I can recall many instances of middle ear tuberculosis healing with improvement in the pulmonary lesion. Three cases were cited to substantiate this.

DR. H. B. GRAHAM, San Francisco, Cal.: The best description of tuberculosis of the ear from a pathologic standpoint was that made about 1913 by Ribbert of the German Otologic Society. The essayist did not mention the fungoid and the gangrenous form of aural tuberculosis. In the latter type the bone is dry and black in color. The bovine form in children is not as dangerous as aural tuberculosis in adults due to the human bacillus. When the infection is all cleared out the child gets entirely well.

DR. H. G. PRESTON, Harrisonburg, Va.: In regard to extrapulmonary tuberculosis, one must keep in mind the relationship of allergy to the organism itself rather than to the invasion of the tissues by the organism.

DR. EDMUND P. FOWLER, New York: We examined from 450 to 500 patients several years ago in a large hospital of tuberculous patients. Our statistics as to the effect on the hearing did not exactly tally with those reported today. It is remarkable how good the hearing was.

7. THE EFFECT OF SEVERE ILLNESS UPON THE HEARING. Edmund Prince Fowler, M. D., New York. (Paper printed in full on p. 388 of the June number, this volume of the ANNALS).

DISCUSSION.

DR. ROBERT WILSON, Charleston, S. C.: There is some question, I think, whether we are justified in assuming that arteriosclerosis may commonly be the determining factor in the hearing loss of old age, because old age is frequently, but not always, accompanied by arteriosclerosis. Also, independently of the presence of arteriosclerosis, it is probable that the capillaries undergo retrogressive changes. This is probably the natural process of growing old. It occurs so gradually that frequently one afflicted does not appreciate its onset and its progress. The ganglion cells are impaired through lack of nutrition from the capillaries. As Dr. Fowler says, the arteriosclerosis is a "patchy disease," different portions of the arteriovascular system being variously involved. The symptoms will thus depend on the organ or organs most affected. We have depended very largely upon eye-ground examination, but if Dr. Fowler's investigation proves fruitful we shall have another.

DR. H. M. TAYLOR, Jacksonville, Fla.: Dr. Fowler emphasizes the importance of changes which may occur in the internal ear as a result of pathologic changes elsewhere in the body. Arteriosclerosis being of a patchy nature, explains why in a group of cases some will have disturbance of the function of the heart, others of the kidneys, others of the ears, eyes, brain, etc. The closest approach to a diffuse generalized arteriosclerosis is found in the blood trunks of the kidneys, while most characteristic patchiness is in the aorta and coronary vessels and in the cerebral vessels. In studying the effect that severe illness may have on the hearing it is important that we do not ignore the possibility of drugs administered being a factor in the production of nerve deafness.

DR. HILL HASTINGS, Los Angeles, Cal.: We all see, occasionally, cases in which there is sudden severe unilateral deafness. I think these cases are due to foci of

infection; the Wassermann reaction is rarely positive. There are also cases of inflammatory lesions of the inner ear, and here the hearing returns. In cases of herpes of the ear there are no ocular manifestations to help us. Therefore, we should be on the lookout for specimens which may show inflammation of the spiral lamina and Scarpa's ganglia.

DR. EDMUND PRINCE FOWLER (closing): I think if we will regularly test old people we may learn some interesting things. The sclerosed vessels are usually found in the internal auditory meatus. We have also found very few Wassermann positive cases. Yet the curve looks like a typical curve of nerve deafness and a specific deafness, because we forget that the person may have syphilis and not have specific deafness.

8. CANCER OF THE LARYNX: OBSERVATIONS FROM TWO HUNDRED CONSECUTIVE CASES. Gabriel Tucker, M. D., Philadelphia, Pa.

A critical review is presented of the last 200 consecutive cases admitted to the Bronchoscopic Clinics at the University and Graduate Hospitals of the University of Pennsylvania. A statistical study as to the probable duration of the lesion is given, also the extent and location of involvement, the classification of the lesion for purposes of treatment, the type of treatment given and the results to the present time. Lantern slides and a moving picture were shown.

DISCUSSION.

DR. CHEVALIER JACKSON, Philadelphia, Pa.: I was very glad to hear the matter of biopsy emphasized so strongly. Biopsy in the larynx is without any danger because of the peculiar lymphatic distribution. One deplorable thing is that statistics of laryngofissure are all cluttered up with cases that do not belong in that category. In lesions involving the anterior commissure the incision should be only through the cartilage, the dissection then elevating the perichondrium, with it the underlying tumor. One should never cut through the growth. The cartilage and its perichondrium are invaded very late, in fact, not until pyogenic infection has paved the way.

Our five-year cures are now 82 per cent. The most appalling fact in laryngeal cancer is that almost all the cases come too late for surgery, even for laryngectomy. We must educate the laity on this point and we ourselves must examine the anterior commissure more definitely.

DR. JOSEPH BECK, Chicago, Ill.: At the Hines Veterans' Hospital cases are given to an expert in irradiation when they are still operable. Thus far I am not convinced, and I still hope for the operation that has been demonstrated here.

DR. PERRY G. GOLDSMITH, Toronto, Canada: In laryngeal cancer the biopsy should be made by someone who is accustomed to accurate laryngeal manipulations, and the pathologic examination should be made by a pathologist experienced in the study of laryngeal lesions. The symptom of hoarseness cannot be emphasized too strongly. Medical students should understand this thoroughly. A negative pathologic diagnosis is dangerous to accept if the clinical case is predominantly malignant. I do not favor radium in the larynx; high voltage may be used in extensive incurable cancer.

DR. W. B. CHAMBERLIN, Cleveland, Ohio: I think we should not only educate the laity but also the medical profession. I wish to ask Dr. Tucker whether he removes the block of tissue with the knife or diathermy.

DR. T. E. CARMODY, Denver, Colo.: I think that five-year cures are important, but some of these cases recur a few years after that period.

I do not think that anyone should use caustics in the larynx, especially silver nitrate.

The endothermic knife is satisfactory in laryngofissure cases, but one must know how to use it properly.

DR. GABRIEL TUCKER (closing): I have not used the endothermic knife. We are afraid of the possible effects on the cartilage.

To find cancer in the larynx after five years does not necessarily mean that it is a recurrence; it may be a new growth.

#### 8. THE BIPLANE FLUOROSCOPE IN BRONCHOSCOPY. Murdock Equen, M. D., Atlanta, Ga.

In his introduction, the author epitomized the history of removing foreign bodies in the air passages with particular emphasis on the American pioneers in this field. The biplane fluoroscope was introduced into bronchoscopy in 1915. During the early days some enthusiastic operators used it as a substitute for bronchoscopic skill with disastrous results. Grier, Jackson and Tucker, aided by Manges, Prendergrass and Pancoast, have improved the technic so that it now entails little risk.

The indications for the use of the biplane fluoroscope are when radiopaque objects in the tracheobronchial tree are unusual, irregularly shaped, with cutting edges or sharp points, or if the foreign body is beyond direct vision through the bronchoscope, either on account of its actual location or on account of associated inflammatory processes. In the esophagus open safety pins, large irregularly shaped objects or those that have been lodged there a long time or that are associated with strictures or other lesions also require the fluoroscope.

Two cases were reported in which the biplane fluoroscope was used. In the first, a child aged six, who had symptoms of pulmonary tuberculosis for nearly two years, a pin was found in the posterior bronchiole of the lower lobe of the left lung. The child made a prompt and uneventful recovery after the removal of the pin. In the second case, a little girl had swallowed a bobbin from a textile machine thirty months before. This had lodged in the upper part of the esophagus and had required a gastrostomy. At the time she came under the author's care, scar tissue around the bobbin had occluded the lumen of the esophagus. Under the biplane fluoroscope



the bobbin was released by means of sharp dissection and extruded through the stomach. Poverty and ignorance prevented the parents from bringing the child back at frequent intervals for dilatations of the esophagus, but more than a year later she was in good condition and able to swallow almost anything.

#### DISCUSSION.

DR. CHEVALIER JACKSON, Philadelphia, Pa.: I heartily endorse all that Dr. Equen has said. The double-plane fluoroscope, while exceptional for certain cases, does not solve all the difficulties. Sometimes the foreign body may be visible on the X-ray film but not under the fluoroscope. It is very important in this work that the proper smaller bronchus be found for the bronchoscope by careful palpation. In the hands of a skillful bronchoscopist, such as Dr. Equen, the biplane fluoroscope is safe and is attended with a large percentage of successes. In the hands of one who has never done eye-guided bronchoscopy and who does it guided by the fluoroscopic screen the mortality is high.

DR. GABRIEL TUCKER, Philadelphia, Pa.: I wish to emphasize that both the roentgenologist and the trained bronchoscopist are necessary for these cases.

DR. RALPH F. DAVIS, Portland, Ore.: I have recently had an interesting case in this connection. A nineteen-months-old boy had a small tack with the head buried, deep in the left lung. In spite of the fluoroscope, we were unable to remove it until the fourth attempt, and then with lower bronchoscopy. Recovery was uneventful.

DR. MURDOCK EQUEN (closing): The biplane fluoroscope is something you may never use, and yet you may need it tomorrow.

#### 10. MYCOTIC INFECTIONS OF THE MAXILLARY SINUSES. H. N. Stevenson, M. D., Rochelle, N. Y.

Seven cases are reported of infection of the maxillary sinus in which fungus forms were found in the pus recovered from these sinuses. These fungus forms closely resembled those of *mucor-histoides*. Six of the cases were classed as chronic sinusitis. Four of these had recurrent infections while under observation, and in two of these cases fungus forms were also found in infections recurring after some months. In five cases the infection was bilateral. The findings indicated that fungi were present as active agents rather than as saprophytes. In one of the cases the sinus infection was associated with acute rheumatic manifestations. The recurrent infection suggested a theory which would account for the production of certain head colds.

#### DISCUSSION.

DR. DUNCAN MACPHERSON, New York: I have had one case of fungus of nasal origin. On looking up the subject, I found that Dr. Stevenson has gathered together about all that is known about the subject. All fungi grow on maltose agar so profusely that differentiation is impossible. From this, however, they may be



transplanted to cornmeal agar which will grow them only sparsely, permitting of easier differentiation.

DR. JAMES G. DWYER, New York: We divide all moulds of the lower grade, leaving out blastomycosis and actinomycosis, into three divisions simply on account of their method of growth. A splendid way to recognize the mucor is to grow it in a moist place. The aspergillus which we get time and again is sexual. The blastomycetes, the yeast, we get only in jars and around injured bones, not as a primary invader. A mould having chromatin is an accidental invader and never pathogenic. There is no difficulty about the classification of moulds, but it is very difficult to determine their pathogenicity.

DR. WILLIAM B. CHAMBERLIN, Cleveland, Ohio: Moulds of the ear are common but they are not easily eradicated. I have had a case recently of a mould in the vestibule of the nose. We finally got rid of it by using phenol-mercuric-nitrate and phenol-mercuric-chloride. We are also using the combination in atrophic rhinitis and for irrigations of the sinuses. We hope to give a report later.

DR. M. A. LISCHKOFF, Pensacola, Fla.: I wish to report a case of a man, sixty-one years of age, who had an aspergillus infection of the nose. We did a sublabial antrotomy, and the fuzzy, white mould could be seen in the curetted membrane. The condition then cleared up.

DR. H. N. STEVENSON (closing): In speaking of chromatin, one means the quality of that particular part of the cell which picks up the stain. This present series of cases was treated principally with metaphen and neosilvol and mercurochrome. I feel this subject warrants further study.

#### 11. POST-OPERATIVE CONDITIONS UPON THE SPHENOID SINUS FOLLOWING ETHMOSPHENOID OPERATIONS. Frederick T. Hill, M. D., Waterville, Me.

The end results of operations on the sphenoid sinus are often unsatisfactory, due to the tendency of the operative opening to contract or even close. This may take place long after cicatrization is complete. Nature tends to replace original structures when possible. This contracture seems to occur more frequently where there is marked asymmetry of the sinuses, in the smaller of the two. Likewise, allergic cases seem more prone to this reformation than the purulent type. Exacerbations of allergic phenomena, previously under control, may follow contracture and again subside when the openings are restored.

#### DISCUSSION.

DR. LYMAN G. RICHARDS, Boston, Mass.: In the execution of surgical procedures on the sphenoid sinus, the operator has one of two main objects in view, dependent on the type of existing pathology. The first is the improvement of drainage in purulent sphenoiditis; the second is the removal of a diseased lining membrane when the latter is felt to be acting as an allergic irritant. The second procedure, I find, necessitates the external approach. Even with this, certain cases may not be successful. Perhaps we must always be prepared to encounter individual tissue reactions just as in the case of the maxillary antrum. As in all surgical work, there lies a happy medium between conservatism and radicalism which must ever be the object of our quest.

DR. HAROLD G. TOBEY, Boston, Mass.: Again we are between the conservative and the radical approach. I agree with Dr. Hill that in the allergic cases when the sphenoid does block up, there is a return of the symptoms. Of course, we all realize that surgery in the allergic cases is oftentimes necessary. A lot of these sphenoids do close up but there are no further symptoms. The question then is, is it always necessary to keep them open?

DR. FRANCIS B. BLACKMORE, Columbus, Ga.: I think that in these cases the biologic approach is probably the most valuable. "Hemoprotein" may be used to lift the infection zone, and "typhoprotein" to lower the allergic zone. Parke-Davis is working this out. Where polyps and grossly infected sinuses are present, surgery is advised.

DR. FRANK KISTNER, Portland, Ore.: I also have had trouble with the sphenoid opening closing, but it is possible to make a plastic from the mucous membrane covering the anterior portion of the sphenoid. In the ideal operation, the lining membrane of the sphenoid is removed entirely. I think it can be done in over 80 per cent of the cases. Then you have the probability of complete, or almost complete, obliteration of the sphenoid with granulation tissue. The closing of the opening in allergic cases may only be due to a temporary allergic edema.

DR. HENRY ROSENBERGER, Cleveland, Ohio: I have recently filled the sphenoids of fifty cadavers with barium paste and had them X-rayed. We were very much surprised at the tremendous size of the sinus. From this study I think the opening closes in those cases with a thick anterior wall. The extreme thinness of the walls adjacent to the optic nerve and trigeminus nerve in many cases would make curettement very dangerous.

DR. FREDERICK T. HILL (closing): The details of technic were left out of the paper. In allergic cases I think the lining of the sphenoid should be removed, hence the external approach. Diathermy has been very unsatisfactory. The internist worked along on all these cases.

## 12. A CLINICAL ANALYSIS OF FIFTY CASES OF CHRONIC SUPPURATIVE OTITIS MEDIA WITH CHOLESTEATOMA. Kenneth M. Day, M. D., Pittsburgh, Pa. (Paper printed in full on page 837 of this issue.)

### DISCUSSION.

DR. SAMUEL J. KOPETZKY, New York: This is a most provocative paper, and in the main I agree with the formulative conclusions the essayist presents. Moisture is a factor, but not the prime factor. The cholesteatoma is a thwarted effort of nature to heal a chronically suppurating ear. Squamous epithelium comes in to form a scar, but because of osteoblastic action by pressure of the foreign body against the bone, irregularity occurs, then as it ingrows, it turns upon itself. Therefore the bone irregularity is a prime factor.

Results are poorer in children because nature has not yet progressed a considerable stage toward cure.

From my experience and observation, I believe all cases of cholesteatoma should be operated on. I also contend that you can dissolve tetrochloride with alcohol, and you can dissolve the detritus, and by keeping the patient under continuous observation you can clean out the collections of detritus, but if you do not smooth out these areas the patient is always a potential subject for an epithelializa-

tion that gradually forms. In the radical operation I even use a magnifying lens to see that the tissue is absolutely smooth so that there will never be any chance for the healing cholesteatoma matrix scar to turn.

DR. JOSEPH C. BECK, Chicago, Ill.: This is the first paper in English since Wittmaack's publication on pneumatization and cholesteatoma formation to show how cholesteatoma reforms. I feel definitely that moisture is the greatest factor in epithelial desquamation. The mechanical treatment outlined has some possibilities of complications unless carried out by such experts as Dr. Day. I believe that a case with hidden cholesteatoma should be operated upon, even in children. However, I do not agree that a matrix is a healthy condition. I like to remove the cholesteatomatous matrix. Smoothness of the cavity is, of course, a very important factor. Lastly, associated bone necrosis must also be removed.

DR. EWING W. DAY, Pittsburgh, Pa.: I have found many cases which could be successfully treated without operation. There are very few men in this country who can do a really good and successful radical operation. I think our cases treated conservatively are as safe as they would have been under the average run-of-the-mill operation.

DR. W. W. PEARSON, Des Moines, Iowa: I have always been conservative in my practice. In regard to the fistula test, I have observed a patient with a dry ear over a number of years and he has never been operated on. I have used the Zeiss telescopic lens for years in my examination of mastoid cavities.

DR. JAMES G. DWYER, New York: I want to congratulate Dr. Day on his conservative, common-sense paper. There are certain cases that must always be operated upon; but the very presence of cholesteatoma in itself does not mean radical mastoid operation.

DR. EDMUND PRINCE FOWLER, New York: I wish to back up what Dr. Dwyer has said. I do not think we get very good hearing after radical operation. These things are especially true in children.

DR. GEORGE L. TOBEY, Boston, Mass.: I think it is very important to emphasize the use of anhydrous solutions. In clinic work I would advise the radical operation; in private practice, I somewhat agree with Dr. Day.

DR. VERNON L. BISHOP, Rochester: I would like to ask why moisture is the primary factor.

DR. KENNETH M. DAY (closing): The lack of moisture implies the lack of infection. Cases with rough places in the bone do not get well and we have to operate. The question of moisture has been known for years, but I have not seen it specified in black and white in the literature that it is a primary factor in the formation of cholesteatoma.

### 13. CYSTS AND RETENTION ABSCESSSES OF THE NASOPHARYNX (Candidate's Thesis). Barney M. Kully, M. D., Omaha, Neb.

Report of eighty-eight cases, mainly from 1880 to 1900, is made. The majority occur between the ages of 18 and 35. Twenty-five per cent are symptom free. In the diagnosis, direct inspection is necessary. Examination with postanasal mirror and nasopharyngoscope is inadequate.

## DISCUSSION.

DR. JOHN B. POTTS, Omaha, Neb.: It is my opinion that these cysts are sufficiently frequent that the routine of Kelley's direct vision adenotome or Yankauer's speculum should be insisted on in all cases in which there is suspicion of focal infection. The only satisfactory way of dealing with these cysts is to remove the anterior wall with biting forceps. If a patient has a headache, postnasal discharge or postnasal scab with constant irritation, or bad breath, and a cyst of this kind is found and taken care of, the symptoms are usually relieved.

DR. JOSEPH C. BECK, Chicago, Ill.: Radiopaque substance may be injected into these cysts and so give the extent of the cavity. Goerke has given us an excellent instrument for operating on these cysts. The electrothermic destruction of the lining membrane is successful and should be mentioned.

DR. FRANCIS B. BLACKMORE, Columbus, Ga.: I wish to demonstrate an instrument in this connection which Dr. Lynch used.

DR. LYMAN G. RICHARDS, Boston, Mass.: I do not think that congenital cysts in the lateral pharyngeal pouch are ever located in the midline.

DR. BARNEY M. KULLY (closing): I wish to emphasize that these cysts are more frequent than we have believed.

## Abstracts of Current Articles.

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### NOSE.

#### **Persistence of Apparent Sinus Pain After Multiple Operations.**

*Skillern, Samuel R. (Philadelphia), Arch. of Otolaryng., 19:415 (April), 1934.*

The author presents the written conception of leading otolaryngologists of the country regarding this problem. A resumé of these opinions is tabulated as well as his own personal findings. Opinions from sixteen rhinologists give the following causes for persistence of pain:

- Incomplete operation, 7.
- Disease elsewhere than in the sinus operated on, 6.
- Systemic disease causing symptoms of sinusitis, 5.
- Synechia or scar tissue blocking drainage or enmeshing the nerve, and painful contractures, 5.
- Direct injury to, or interference with, a nerve, 4.
- Operation on the ethmoids and turbinates for insufficient reason, 4.
- Individual nervous reaction to pain, 3.
- Neuritis of the fifth cranial nerve, 3.
- Atypical neuralgia of unknown origin, 3.
- Reaction to insult of the periosteum and bone, especially surrounding the foramen, 3.
- Injury to the sympathetic plexus, 2.
- Diversion of normal air currents within the nose, 1.

TOBEY.

#### **Atrophic Rhinitis.**

*Adam, James (Glasgow), J. of Laryng. and Otol., 49:375 (June), 1934.*

The author reviews a series of 141 cases of atrophic rhinitis in which at least 78 per cent began before puberty, and about 42 per cent during the first seven years of life. These are the years during which the face, nose and its accessory cavities are normally developing most quickly. This development is consequently retarded. When the disease begins in adult life, as it rarely does, the typical facies

of atrophic rhinitis does not occur. The disease begins as inflammation of the nasal mucosa and in at least half the cases, probably over 66 per cent, there is sinusitis. This tends to keep the inflammation alive and is often missed. The ethmoids and also the adenoids are more often affected than is realized. The present custom of dealing with adenoids early has reduced the incidence of atrophic rhinitis. Another factor is deficiency in vitamins, especially vitamin A. This deficiency impairs defense against infection and lowers endocrine function. It may also impair nervous function.

The mucosa reacts first by hyperplasia; later, owing to fibrosis, there is glandular atrophy and change from columnar epithelium to stratified epithelium; but if the sinusitis is conquered early enough by proper surgical and other measures there may be considerable recovery with disappearance of crusts and fetor. The bone of the thin lamellæ of the turbinates and the ethmoid cells reacts by atrophy, that of the walls of the accessory cavities by sclerotic thickening. Similar sclerosis is found in the mastoid process of children with chronic suppuration of the middle ear. The paranasal sinuses may fail to reach their full development with consequent facial modification.

GOLDSMITH.

#### **The Innervation and Vascular Supply of the Antrum.**

*Strong, Cecil (Birmingham), J. of Laryngol. and Otol., 49:400 (June), 1934.*

This paper is a report of research into the condition of the teeth after the operation of radical antrotomy. Some uncertainty has existed as to whether the teeth recover their sensation, remain insensitive, or are dead. An attempt was made to investigate the condition of the pulp of the teeth, immediately below the long incision, and to obtain information regarding the vascular supply of that neighborhood. The investigation is divided into two parts: (1) A clinical examination of the patients' teeth in thirty-three cases after this operation, and (2) determination of the courses of the nerve canals to individual teeth and their vascular supply.

The clinical part of the investigation consisted of examining the teeth of patients after operation by transillumination, percussion, thermal tests, Faradic current and radiography. Testing by the application of ice was found to be most reliable. The striking fact about these teeth is that, were it not for this test, they would have been passed as normal. In all cases examined, there was this anesthesia in at least one, and sometimes in three teeth, but otherwise they showed

no sign that there was anything wrong with them. The number of teeth which suffer depends upon the size and position of the antral opening and quite a small opening will cause the loss of sensation in one tooth only.

The second point of the investigation was carried out by injecting the superior maxillæ with lipiodol and examining them by X-ray. They showed vascular anastomoses along the antral floor and the possibility of injecting the canals subservient to several teeth by injecting one tooth socket. Microscopic sections of this region were prepared to show nerves and blood spaces. The size of the vessels is such that a collateral circulation more than sufficient to keep a tooth alive could be established. The dental nerves run in bundles and do not ramify among the vessels. There appears to be a vascular plexus but not a nervous one.

In one case a denervated tooth was extracted and the pulp examined by serious transverse sections. It was impossible to find any areas of degeneration, with the single exception of a few nerve filaments, which were known to have been cut.

From these investigations it appears that the teeth are denervated but not devitalized by the trauma to the anterior antral wall above their apices. Their blood supply is still present, and probably comes through a collateral anastomosis, along the antral floor and partly through the antral mucosa.

GOLDSMITH.

**The Effect of Radical Antral Surgery on Bronchitic Asthma.**

Warner, W. P., and McGregor, Gregor (Toronto, Canada), *J. of Laryng. and Otol.*, 48:595 (Sept.), 1933.

Thirty-one cases of bronchitic asthma were treated by radical antral operation, which included the removal of the thickened mucoperiosteum. They were followed closely for from six months to two and a half years, and in two cases only were the results decidedly favorable and permanent. All cases had a period of freedom from asthma following the operation but relapsed later. The longest period of freedom was twenty-seven months, the shortest two weeks, and the average of all cases four months. These results are so poor that radical antral surgery undertaken for indications as outlined above should be recommended with a great deal of hesitation.

There were no peculiarities in those cases apparently benefited for a long time, which would enable one to decide for certain on which case to operate. The cases apparently benefited had had asthma

for a relatively short time and presented markedly thickened mucoperiosteum.

Results of the treatment of asthma by operative procedures on the nose should not be given until the patients have been observed for a long period of time following the operation. All cases but one ceased to have asthma for two weeks following the operation. This fact led to the operation being performed as a life-saving measure in one instance.

Five cases of chronic bronchitis were treated by radical removal of thickened mucoperiosteum and were observed for a period of two years. No benefit was seen to be obtained as a result of the operation.

GOLDSMITH.

**Consideration on the Origin of Congenital Fistula on the Dorsum Nasi (Considerazioni sull'origine delle fistole congenite del dorso del naso).**

*Simonetta, B. (Pisa), Boll. delle Malatt. Orecch. Gola. Naso.* 60:503 (December), 1933.

The writer briefly presents a case of a congenital fistula located at the midline of the dorsum nasi, in a child, three years old. Histologic description of the tissue is presented and from the study of the specimen he reviews critically the various theories advanced to date as to the genesis of this rare deformity. He proposes that these fistulae are due to alteration in the closing process of the anterior neuropore and concludes by explaining the various elements that support his theory.

SCIARRETTA.

**Radiologic Demonstration of the Cerebral Fluid Passing Through the Nasal Mucosa (Dimonstazione radiografica della scarico del—liquor—attraverso la mucosa nasale).**

*Bronzini, A., Riv. Oto-Neuro-Oftal. and Rad-Neuro. Cbir.,* 10:577 (September and October), 1933.

The writer injected into the cisterna magna of living animals (dogs under anesthesia) a radiopaque substance (iodide of sodium, potassium, lithium, bromide or especially uroselectan) and made radiographs every minute. He noticed that one minute after the injection of the radiopaque fluid the opacity of the nasal mucosa began to increase and reached its maximum at the tenth minute and it disappeared completely in fifteen minutes. This proves the rapid diffusion of these fluids to the nasal mucosa and presumably the rapid discharge of the cerebral fluid through this route. He prefers dialyzed india ink, previously used in his experiments, to this new method. Seven X-ray photographs accompany the article.

SCIARRETTA.



**Some Considerations About Acute Suppurative Frontal Sinusitis: Four Fulminating Cases.**

McNally, W. J. (Montreal). *Trans. Am. Laryng. Assoc.*, 1934.

Four cases are presented of acute fulminating frontal sinusitis in young adults. They were given prompt, simple internal drainage. The X-rays of the sinuses were misleading in three of the cases. In at least three of the cases the organism was staphylococcus pyogenes aureus. On admission the temperature ranged from 101 to 104 degrees F. They developed complications, two having frontal lobe abscesses and two osteomyelitis of the skull. All four cases recovered.

IMPERATORI.

**Nasal Polyposis Invading All the Paranasal Sinuses (Poliposi Nasale Diffusa a tutti i seni della faccia).**

Ricci, B. (Firenze), *Boll. delle Malatt. Orecch. Gola. Naso.* 51:463 (November), 1933.

The author describes a case of nasal polyposis, in a man 70 years old, discovered at autopsy, whose death was due to an acute bilateral pulmonary edema. All the paranasal sinuses were completely filled with multiple polypi, the space between them with purulent exudate. The internal wall of the frontal sinus in some places was destroyed, permitting septic material to pass through, producing a circumscribed pachymeningitis. He quotes the literature and considers the case reported by Chamberlain in the Laryngoscope (1913) the only one on record to be similar to his.

SCIARRETTA.

**Can Ozena Be Cured by Arrest of Nasal Respiration? Case Report (Guérison de l'ozéne par suppression de la respiration nasale?)**

Vasiliu and Criscota (Bucarest), *Rev. d'Oto-Neuro-Oph.*, 12:276 (Apr.), 1934.

A girl, twenty-four, with foul obstructive ozenal crusts in the nose, pharynx and larynx, was tracheotomized because of acute dyspnea due to crusts accumulating on some laryngeal papillomas. For a month thereafter, until decannulized, the nose and throat were free from crusts or odor; the improvement has remained for eight months.

Experiments by tightly plugging one or both nostrils of other ozena cases for twenty-four hours similarly showed disappearance of crusts and diminution of odor; but the nostrils became irritated after forty-eight hours and plugging was discontinued. Crusts returned in a few days after these short experiments. Evidently a long period of physiologic rest is essential. No cleansing or antiseptic agents were used except to take out crusts once, before beginning the treatment.

FENTON.

**Treatment of Ozena by Cholin Salts (Les sels de choline dans le traitement de l'ozéne).**

Guns, P. (Louvain). *Rev. de Lar., Ot., Rhin.*, 55:367 (Mar.), 1934.

Reporting on five years' use of acetylcholin and the more stable hydrochloride of carbaminoylcholin, Guns remains most favorably impressed with their powers of parasympathetic stimulation. He has quit using ointments or oily suspensions and now secures free watery discharge from the nose, disappearance of crusting and odor, and apparent thickening of the turbinal mucosa, using a solution of eight to ten milligrams of carbaminoylcholin dissolved in twenty cc. of water, ten drops three times daily in each nostril for two weeks. This is repeated each month until permanent results seem to have arrived.

FENTON.

**Intranasal Mucous Membrane Color Changes as a Guide to the Status of the Sympathetic and Parasympathetic.**

Jarvis, D. C. (Barre, Vt.). *Trans. Am. Laryng. Assoc.*, 1934.

It has been difficult to make clinical application of knowledge relating to the autonomic nervous system because of lack of a suitable index to be used at time of contact with the patient. Characteristics of sympathetic division; characteristics of parasympathetic division are antagonistic. The author reviews the general considerations of intranasal mucous membrane color changes and the characteristics associated with the pale mucous membrane, with an interpretation of red and pale mucous membrane characteristics. He discusses the foundation on which mucous membrane color changes rest and the application of the foregoing to treatment.

IMPERATORI.

**PHARYNX.**

**Concerning the Peritonsillar Spaces: An Anatomic Study.**

Wood, George B. (Philadelphia). *Trans. Am. Laryng. Assoc.*, 1934.

By the injection of a gelatin mass into various areas around the faucial tonsil certain anatomic relations were demonstrated, these pertaining to the fascial planes and to the attachment of the muscles and the contillar capsula. It was shown that the tonsil is easily separated from the superior constrictor muscle; that there are definite space limitations apparently brought about by the position of the faucial and pharyngeal muscles and the perimuscular fascia; that as the muscles of the palate and faucial region run mostly in a longitudinal direction the extension of the injected material is limited

much more in its anteroposterior extension than in its upward or downward extension; that an injection in front of the palatoglossal muscle does not extend backward; in the tonsillar fossa it extends readily upward and downward but not between the tonsil and the posterior pillar; in the posterior pillar it does not reach the tonsillar fossa but extends upward on the posterior surface of the palate and comes in intimate contact with the posterior surface of the tonsil; and finally in the lateral pharyngeal wall it extends upward to the eustachian tube and to an unlimited extent downward but does not pass into the posterior pharyngeal wall or into the posterior faucial pillar.

IMPERATORI.

**Motor Innervation of the Soft Palate (L'innervation motrice du voile du palais).**

Fagart, M. (Bordeaux). *Rev. de Lar., Ot., Rbin.*, 55:451 (Apr.), 1934.

After citing the disagreements existing among anatomists respecting the participation of the facial in palate innervation, Fagart reviews eleven cases of undeveloped petrous, of which seven had facial palsy; and of the seven, four had unilateral palatal palsy.

Experimentally he was able to produce this paralysis in a number of rabbits by sectioning the facial above the geniculate ganglion. He thus concludes that more or less of the palatal motor innervation may be supplied via the facial.

FENTON.

**LARYNX.**

**Malignant Disease of the Bronchus.**

Ormerod, F. C. (London), *J. of Laryngol. and Otol.*, 48:733 (Nov.), 1933.

The author reviews a series of twenty-seven cases of malignant disease of the bronchus, the large majority of which occurred in the male sex. He gives a description of the symptoms, clinical signs and radiologic examinations before and after the injection of lipiodol into the bronchial tree. The cases were examined by bronchoscopy and the diagnosis confirmed by biopsy. Several X-ray illustrations are given showing that a growth in the bronchus is almost always convex, while a fusiform narrowing is probably due to chronic inflammation. The number of cases of this disease correctly diagnosed during life has greatly increased in the last ten years, which is due to a large extent to the improved methods of examination, such as radiography and bronchoscopy, and also to physicians' familiarity with cancer of the lung.

Carcinoma of the bronchus is frequently of the squamous-celled variety and the writer suggests that all the different types of carcinoma arise from the layer of small ovoid cells which support the layer of ciliated columnar cells. In their pathologic studies they have not seen any section which gave the impression that the ciliated columnar cells of the bronchus or the flattened epithelial cells of the alveoli could give rise to the growth. The squamous-celled type tends to occur in older people, the columnar and ovoid types being found among the younger. Metastases occur commonly in the mediastinal glands, from which the tumor may invade the hilus of the opposite lung. They also occur commonly in the suprarenal gland, liver, pancreas, brain and almost any organ. A tumor may progress through the lung tissue to the pleura, and then the whole pleural surface may become studded with secondary deposits. Such cases are sometimes described as primary tumors of the pleura but really have a bronchial origin.

The ideal treatment of a growth in the bronchi is complete removal. Lobectomy is carried out in selected cases and encouraging results have been obtained, but the selection of suitable cases presents some difficulty. The majority of cases under review were treated by the application of radium in the form of emanation. Radon seeds are used and may be applied either through the bronchoscope or through the chest wall after thoracotomy. Treatment by the radium bomb has been very disappointing. The distance from a growth in the lung to the radium on a bomb is too great with any of the existing amounts of radium. Deep X-ray therapy has been slightly more encouraging and one or two cases, especially in the upper lobe, showed some improvement. The results of treatment of carcinoma of the bronchus are not very satisfactory, owing to the advanced stage in which most cases are seen.

GOLDSMITH.

*The Presence of Intra-epithelial Capillaries in Laryngeal Papilloma (Sulla presenza di vasi intra-epiteliali nei papillomi laringei).*

Serge, R. (*Torino*), *Boll. delle Malatt. Orecch. Gola. Naso*, 51:453 (November), 1933.

The author reports the histologic findings in three cases of laryngeal papilloma. He discusses the subject thoroughly, reviewing the literature. The two photomicrographs in the article illustrate clearly how the capillaries are surrounded by epithelial cells and only a single layer of endothelium separates the epithelium from the blood stream.

SCIARRETTA.

**The Lymphatic System in Relation to Recurrent Laryngeal Nerve Paralysis Secondary to Cancer of the Breast.**

Schwartz, H. W. (Halifax, Canada), *J. of Laryngol. and Otol.*, 49:221 (Apr.), 1934.

The writer reports a case of recurrent laryngeal paralysis, secondary to cancer of the breast, and reviews the sixteen cases previously reported in the literature. The explanation of this phenomenon, put forward by Dr. A. Logan Turner in his original article in 1921 and which has prompted all subsequent reports, is examined and questioned in the light of more recent knowledge of the anatomy of the lymphatic drainage of the breast and its connections.

The conclusion arrived at is that the explanation suggested is subject to correction in its essential points. The writer is inclined to believe that in the majority of cases the infection travels via the substernal route or by way of the axilla and infraclavicular glands to reach the internal jugular chain, which in turn has, as an afferent, the paratracheal distribution.

GOLDSMITH.

**Associated Paralysis of the Vocal Cord.**

Burger, H. (Amsterdam), *J. of Laryng. and Otol.*, 49:1 (Jan.), 1934.

This paper discusses the pneumogastric nerve in its relation to the neighboring cranial nerves. Since Hughlings Jackson described in 1864 a case of paralysis of the last three cranial nerves, a number of other combinations have been reported, which have all been named after medical men. The writer reviews these syndromes and questions whether the classification is justified. It is pointed out that we should refrain from putting up a series of "syndromes" on the basis of accidental combinations, even though these syndromes bear the names of highly distinguished medical men, because such names only lead to confusion.

The study of the associated paralysis of the vocal cord has contributed substantially to our knowledge of a number of problems. After an anatomic and clinical study which is illustrated with diagrams, the author suggests that the associated paralysis of the vocal cord be reduced to the four following syndromes: (1) Syndrome of the bulbar nerves; (2) syndrome of the jugular foramen; (3) syndrome of the parapharyngeal space; (4) vocal cord-diaphragm syndrome.

GOLDSMITH.

**Radiologic Investigation in Tumors of the Larynx and Pharynx (L'Indagine radiologica nei tumori della laringe e della faringe).**

Piccio, C. (Milan), *Arch. Ital. di Otol., Rino. E Laryng.*, 44:641 (November), 1933.

The author writes a thirty-page article reviewing the literature superficially, giving the technic in detail, and discussing, from the radiologic point of view, the variations and extent of calcification and ossification processes of the laryngeal cartilages. The article contains nineteen roentgenographs with corresponding sketches, explaining thoroughly the normal and pathologic structures. He gives the history and endoscopic findings of a number of cases. He further discusses the importance of considering irregularities and variations of the cartilages obscuring certain areas, interfering in diagnosing pathologic conditions. Experience in reading these X-ray plates will clearly show the extent and location of a neoplasm, destruction of the cartilages and edema, or deformities resulting from edema. He considers the radiographic examination of the larynx, pharynx and upper portion of the trachea absolutely essential when surgery is to be employed, because it explains, clarifies, confirms and at times discovers conditions not visible by either direct or indirect laryngoscopy. Moreover, it should be used to advantage when endoscopic manipulations are impossible.

SCIARRETTA.

**Indications for and Results After Total Laryngectomy (Indicações e resultados da laringectomia total).**

Porto, G. (Campinas, Brazil), *Rev. Oto-Lar. de S. Paulo*, 2:107 (Mar.-Apr.), 1934.

Reviewing the literature and citing his own fifteen cases of intrinsic cancer with 66 per cent cured for more than three years. His operative mortality was 6 per cent. Porto advises against radiation therapy. Surgery is his method of choice; laryngofissure is reserved for circumscribed growths not passing the median line. Rapid growth in a patient under thirty-five suggests laryngectomy, even if the lesion is limited to one cord. Also he is inclined to agree with some European operators that occasionally operation on a cancer of recent origin will be more likely to be followed by recurrences or metastases than will delayed operation on a growth of long standing. The latter, providing the growth has remained intrinsic, seems to have fostered relative immunity to the slowly growing cancer cells.

FENTON.

**Treatment of Laryngeal Tuberculosis by Collapse Therapy Through Alcohol Injection of One Recurrent Nerve (Essai de traitement de tuberculose du larynx par collapsothérapie hémilaryngée provoquée par alcoolisation du nerf récurrent.**

Prof. Vernieuwe (Ghent), *Rev. de Lar., Ot., Rbin.*, 55:341 (Mar.), 1934.

The author reports paralysis of the left recurrent nerve secured by injection of one drop of 98 per cent alcohol into the nerve sheath exposed by surgical dissection; this paralysis lasted three months. During this time tubercular ulceration and pale edema of the left cord healed completely, and hoarseness and dysphagia disappeared.

Usefulness of this method is limited strictly to beginning unilateral lesions without perichondritis or interarytenoid involvement, and to persons already hoarse and dysphagic.

FENTON.

**Paralysis of the Larynx Due to Lead Poisoning.**

Myerson, M. C. (New York). *Trans. Am. Laryng. Assoc.*, 1934.

Involvement of the larynx due to lead poisoning is a rare condition. The literature on this subject is reviewed and all reported cases are tabulated. A recent experience is added, making a total of twenty cases of paralysis of the larynx due to lead intoxication. The occurrence of interarytenoideus and cricothyroid and adductor paralysis in this series would seem to challenge the validity of Semon's law. In several instances the paralysis disappeared after appropriate therapy. In paralysis of the larynx of obscure etiology, the possibility of lead intoxication should be considered.

IMPERATORI.

**EAR.**

**The Leaking Brain Abscess.**

McKenzie, Dan. (London), *J. of Laryng. and Otol.*, 48:797 (Dec.), 1933.

In this article the author reviews thirteen cases in which the cause of persistent suppuration from the ear was found to be a spontaneous rupture and evacuation of a brain abscess. The brain abscess which, rupturing into the ventricles or meningeal spaces, floods the cerebrospinal system with virulent pus and leads to death in a few hours, is well known but what this paper deals with is the spontaneous rupture and gradual discharge of a brain abscess, whether internally or externally, in such a manner as the title indicates.

There is considerable variety to the effect of leakage upon the clinical aspect of brain abscess. In one case the abscess formed, ruptured externally and evacuated itself, without attracting any atten-



tion whatever. It is of course impossible to say how often a brain abscess so entirely latent occurs. The leakage occasionally prevents the usual increase in intracranial pressure with all its dangers. In most of the cases recorded, however, not only was the external discharge remarked and watched, but increasing intracranial pressure, ultimately necessitating operation on the brain.

The onset of the discharge is usually sudden and coincides with relief of pain and headache. It is generally copious and persistent, the quantity being apparently greater than can be produced by the mastoid and middle ear spaces alone. The pus composing it is thick and not infrequently fetid. Although leakage of a brain abscess seldom leads to spontaneous cure, it tends to moderate symptoms and to retard the progress toward fatal issue.

The differential diagnosis between a large leaking extradural abscess and a brain abscess will often be difficult, and may be impossible, until the patient is on the operating table, unless brain detritus is discovered in the pus. In any case, the dural covering of all extradural abscesses should be carefully examined at operation.

The treatment as to the time and place where the surgeon should seek to drain the abscess is discussed in detail. The most favorable time for draining the abscess may depend upon the actual quantity of pus discharged, but more will depend upon the patient's general condition and particular symptoms. In the operation the surgeon should attempt to offer sufficient drainage to the abscess without too rashly encroaching upon the adjoining meninges and brain with the risk of spreading the infection.

GOLDSMITH.

**Secondary Thiersch Grafting of the Radical Mastoid Cavity Through the Meatus.**

Daggett, W. I., and Bateman, G. G. (*London*), *J. of Laryng. and Otol.*, 49:169 (Mar.), 1934.

In this article the authors recommend a return to the method of Thiersch grafting through the meatus fourteen days after the original operation. They prefer for several reasons this method to that of grafting at the completion of the original operation or of reopening the wound later to put the grafts in place.

The important points in the technic are refashioning a wide meatus permitting an easy insertion of the grafts, using intravenously evipan sodium as an anesthetic for the correct placing of the grafts, and filling the cavity with a special wax in order to keep the grafts in position.

GOLDSMITH.



**Improved Operative Technic for Suppuration of the Petrous Apex.**

Myerson, M. C., Rubin, H. W., and Gilbert, J. G. (New York). *Arch. of Otolaryng.*, 19:699 (June), 1934.

These authors propose to modify Eagleton's procedure, simplifying his technic and drawing attention to more landmarks. Illustrations are given and the operative procedure described.

The approach is through the anterior surface (called superior by most authors) of the petrous pyramid after elevating the temporal lobe of the cerebrum out of the way.

The evolution of the technic is based on a detailed anatomic study of 200 bones in 100 autopsies and twice on living subjects.

TOBEY.

**Labyrinthine Tests and Their Aid to Diagnosis.**

Tweedie, Alex. R. (Nottingham), *J. of Laryng. and Otol.*, 49:160 (Mar.), 1934.

The author discusses the value of the labyrinthine tests for vertigo and stresses the importance of the caloric tests and the galvanic tests compared with those of rotation. Vestibular nystagmus is a question of the interaction of central influence on uncontrollable deviation. There are three underlying factors in nystagmus: (1) The initial deviation; (2) the secondary involuntary reflex, and (3) the true volitional effort.

The clinical application of the labyrinthine tests is reviewed, and it is pointed out that the correct interpretation is often difficult because more is known of physiologic results to artificial stimuli than of pathologic lesions. Pathologic disturbances of sensory organs produce an alteration of the normal subjective sensation, as for instance, in lesions of the olfactory nerve or the retina.

GOLDSMITH.

**Radiologic Demonstration of an Otogenous Cerebral Abscess of the Right Temporal lobe (La dimostrazione radiografica di un ascesso cerebrale otogeno del lobo temporale destro).**

Calderoli, I., and Gavazzoni, A. (Bergamo), *Riv. Oto-Neuro-Oftal. and Rad. Neuro. Chir.*, 10:505 (September and October), 1933.

The authors review the literature which concerns the introduction of the various contrast media into abscess cavities of the cerebrum for roentgen examination. These substances are considered harmless by most workers. The writers have used gauze saturated with a 40 per cent solution of abrodil (Bayer) into a large abscess of the right

temporal lobe without the slightest reaction and the healing proceeded normally. They briefly present a case and also four radiographs demonstrating clearly the morphology, the size and the exact location of the main sac; and they further show an air pocket located postero-superior to the opaque substance, thus indicating the extent of the cavity. They found this method useful in judging the healing progress.

SCIARRETTA.

**Laterocervical Fistula from Osteitis of the Cranial Base in a Patient Affected by Tubercular Mastoiditis (Fistola latero-cervicale da osteite della base cranica in individuo affetto da mastoidite specifica).**

*Antognoli, G. C. (Rome), Valsalva, 9:834 (November), 1933.*

The author describes a case of tuberculous necrosis at the apex of the right petrous pyramid, following homolateral mastoiditis, producing an ossifluent abscess, which, with a tortuous course, made its exit at the right side of the neck posteriorly to the sterno-cleido-mastoid muscle. He brings out the importance of radiographic examination with the aid of lipiodol injected into the neck fistula and of a thorough anamnesis for making a correct diagnosis; also the rarity of this type of cases and the efficacy of injecting Beck's paste, of ultraviolet rays and of intravenous iodine medication.

SCIARRETTA.

**Clinical Report on Sinus Thromboses of Otitic Origin (Étude clinique des thromboses des sinus d'origine otique).**

*Eliasson, M. (Leningrad). Rev. de Lar., Ot., Rbin., 55:203 (Feb.), 1934.*

Reviewing 121 cases of otitic thrombosis in Prof. Levin's clinic, from 1925 to 1932, statistics are as follows: Mortality, 36.3 per cent; 42 per cent of men, 28.8 per cent of women; excluding moribund cases and those with other manifest conditions on arrival (brain abscess, meningitis, cavernous thrombosis), mortality drops to 23.1 per cent.

Infection penetrated by contiguity (osteitis) in 86.3 per cent; in the remaining 13.7 per cent, bone and sinus were unaltered in six cases; bone undamaged, sinus altered in four cases; bone altered, sinus undamaged, six cases.

All cases without fever progressed favorably. Ten per cent of meningeal symptoms were an unexpectedly grave factor in otherwise uncomplicated thromboses. Pulmonary metastases were most frequent (sixteen out of thirty-eight cases) with the highest mor-

tal. Mortality from metastases was far higher in chronic (60 per cent) than acute (27.7 per cent) cases.

A few cases had no ear symptoms and were hard to diagnose. Two cases (1.7 per cent) with the usually benign central perforation of the membrana tympani went on to thrombosis. The most certain sign of thrombosis is deep tenderness over the upper part of the jugular vein (Levin's symptom).

In the Leningrad otologic service, mastoidectomy and treatment of the thrombus are done generally in one stage (70.3 per cent). Ligation is frequent, but only when indicated: results, 68.4 per cent cures after ligation; 45.5 per cent cures when ligation was not done. Cases with meningeal symptoms have a bad prognosis. Death was caused by meningitis, 47.5 per cent; septicemia, 43.5 per cent; other causes (brain abscess, carotid hemorrhage), 9 per cent.

FENTON.

**Tumors of the Ear (Contribution à l'étude des tumeurs de l'oreille).**

Alonso, Prof. J. M. (Montevideo). *Rev. de Lar., Ot., Rbin.*, 55:409 (Apr.), 1934.

This fine forty-two page review of 268 cases in the literature since 1900, with the author's reports of fifty personal observations, presents an excellent classification following Manasse, and well-justified methods of treatment.

FENTON.

**Vertigo.**

Brain, W. Russell (London), *J. of Laryng. and Otol.*, 49:153 (Mar.), 1934.

In this article the writer discusses aural vertigo and its differentiation from vertigo due to lesions elsewhere. A subjective sense of rotation, either of the patient or of his surroundings, is the commonest form of aural vertigo but it is by no means always present. Vertigo is defined as the sensation of a disordered orientation of the body in space. It is because it is the sensory expression of a disorder of function which can be produced in many ways that difficulty in estimating the significance and diagnosing the source of vertigo arises.

The pathogenesis of aural vertigo is in some cases easy to understand, but there are a large group of individuals suffering from "Ménière's syndrome" in which the pathology remains obscure. It is suggested that there is a condition of the labyrinth equivalent to papilledema, and many forms of retinitis seen in the fundus oculi. The venous drainage of the labyrinth is even more intimately related to the intracranial venous circulation than is that of the eye. There is probably no feature which is absolutely pathognomonic of

aural vertigo but certain features which in many cases enable one to recognize it are reviewed. Attention is drawn to the points by which vertigo due to lesions of the eighth nerve differ from those due to a lesion of the pons and medulla, intracranial tumors, epilepsy and other conditions. In the medical treatment of aural vertigo, the writer has found luminal to be the most satisfactory drug. Small doses will often make a patient comfortable and larger doses can be given, if necessary subcutaneously, during a severe attack.

GOLDSMITH.

**Fatal Complications of Otitis Media, with Particular Reference to the Intracranial Lesions in a Series of Ten Thousand Autopsies.**

Courville, C. B., and Nielson, J. M. (*Los Angeles*), *Arch. of Otolaryng.*, 19:451 (April), 1934.

The protocols of 10,000 autopsies have been reviewed with special attention having been given to the intracranial complications of otitis media and mastoiditis.

Difficulties in evaluating autopsy records are discussed. Death in cases of otitis media is usually due to an associated malnutrition or dehydration, consequential bronchopneumonia or diarrhea, septicemia or bacteremia or an intracranial complication. The highest death rate is in the first year of life when intracranial complications are less common.

Infection in the petrous pyramid has been studied with particular attention to the intracranial complications; subdural abscesses and dura fistula are discussed. The incidence and pathogenesis of thrombosis of the venous channels and consequent complications; chronic arachnitis and cerebral and cerebellar abscesses are discussed.

A method for removal and study of the brain postmortem is suggested.

TOBEY.

**Blood Infection from Otitis Media.**

Glass, E. J. Gilroy (*Nottingham*), *J. of Laryng. and Otol.*, 48:754 (Nov.), 1933.

An analysis of sixty-three cases of blood infection from otitis media is presented with the object of studying the prognosis and the results of treatment and of determining the general factors of the infection. A bacteriologic examination of the blood was made in most of the cases, but in some the diagnosis was based on clinical grounds alone. In none, however, was there any reasonable doubt that a blood infection existed, and the mortality rate in the two groups was almost identical. The cases reviewed are presented in tabular form and a summary of five illustrated cases of special interest are given.

It was found that the following groups have a relatively low mortality rate: Children under ten years—Cases of otitis media operated upon during the first week; cases developing metastatic abscesses; hemolytic streptococcal infections treated with antiscarlet fever serum.

The prognosis was bad: In patients over forty years of age; in chronic suppurative otitis media and acute exacerbation; if septicemia has developed before operation; in cases showing subcutaneous hemorrhage or jaundice.

GOLDSMITH.

**Function of the Utriculo-endolymphatic Valve: Two Cases of Ruptured Saccules in Children.**

*Bast, T. H. (Madison, Wis.), Arch. of Otolaryng., 19:537 (May), 1934.*

This valve has been recognized since 1928 but its function not determined. There are clinical cases on record where trauma produced deafness but did not at the same time interfere with the function of the static labyrinth.

Two cases reported here showed the cochlea duct and saccule collapsed due to rupture of the saccule, but the utricle and semicircular canals were normally distended.

These two cases showed that the auditory division (cochlear duct and saccule) of the endolymphatic system may be disturbed to the extent of collapse without damage or collapse of the vestibular division (utricle and semicircular canals). These cases further indicate that the "utricle-endolymphatic valve" is responsible for the maintenance of apparently normal pressure in the utricle and semicircular canals when the pressure in the saccule and cochlear duct is suddenly reduced.

TOBEY.

**MISCELLANEOUS.**

**Treatment and Management of Nontuberculous Pulmonary Abscess, with Special Reference to a Series of Twenty-five Consecutive Cases.**

*Cummings, Geo. O. (Portland, Me.), Arch. of Otolaryng., 19:684 (June), 1934.*

The author discusses treatment of this condition and presents twenty-five cases under his own care. He concludes that the earlier adequate treatment is instituted after the onset of the disease, the greater is the hope for the recovery of the patient.

The type of therapy must be suited to the particular case. Basic medical treatment is fundamental, whatever other type of therapy is employed; sodium perborate should be prescribed as a dentifrice as a routine; postural drainage is of value at some time in the man-

agement of almost every case; inhalations of carbon dioxide should be borne in mind; transfusions play an important part in supportive therapy; vaccines are worthy of trial; nonspecific proteins do not seem logical; arsphenamins should be used if the organisms of Vincent's angina are found in the sputum; emetin hydrochloride should be administered if *endamoeba histolytica* is found to be the offending organism; specific serums do not seem logical; artificial pneumothorax is of real worth in the treatment of parenchymal and central lesions and in the treatment of hemorrhages arising in the course of this disease; physical therapy is of tonic and psychic value.

Bronchoscopic aspiration is the most valuable form of therapy, second to postural drainage, in the treatment of this condition. It is also of value in differential diagnosis and in conjunction with roentgen rays in the localization of the abscess.

Phrenicectomy may be used in the treatment of parenchymal abscesses of the lower lobes and may be employed as an aid in collapsing the lung by artificial pneumothorax; thoracoplasty is of worth in the treatment of soft-walled parenchymal abscesses that are draining well by the tracheobronchial tree; extrapleural pneumolysis is a modification of thoracoplasty; intrapleural pneumolysis may prove of value in conjunction with artificial pneumothorax; incision with drainage is the treatment par excellence for peripheral abscesses; lobectomy is better adapted to the treatment of unilobular bronchiectasis.

TOBEY.

#### **Abscess of the Brain.**

*MacKenzie, K. W. (Ipswich), J. of Laryng. and Otol., 49:357 (June), 1934.*

Cases of brain abscess occur so rarely that an aural surgeon has a rather small experience with the disease, and it is only by studying and comparing the results and methods of others that any advancement in treatment can be achieved.

The writer reports eight cases of brain abscess, six of which were cured, a recovery rate of 75 per cent. Seven of the cases were the result of chronic suppurative otitis media. Four of these cases were temporosphenoidal lobe abscesses, two of which recovered; and three were cerebellar abscesses, all of which recovered. He points out that his treatment has not been strictly orthodox, as he postponed operation until pressure symptoms became well marked, by which time a local immunity had developed. Furthermore, delay renders the discovery and drainage of the abscess very easy, and also results in an immediate hernia of the brain on the incision of the dura which, by compressing the dura against the bony margins of the tre-

phine opening lessens the risk of the onset of meningitis. A "delayed" operation is necessary for success, and drainage through a large trephine wound, the writer believes, is more likely to be successful than drainage through the mastoid alone. The eight cases and their treatment are reported in detail.

GOLDSMITH.

**A Method of Specific Treatment in Certain Streptococcic Infections.**

*Baum, Harry L. (Denver). Trans. Am. Laryng. Assoc., 1934.*

The paper describes a method of using therapeutically immune serum taken from patients recently recovered from scarlet fever and other diseases of streptococcic origin. Such serum has been found to be specific in the treatment of a variety of streptococcic infections, even though the disease treated has no clinical relationship to the disease following which the serum has been taken.

The donor is bled, the serum separated, ampouled, and stored ready for use when needed. Specificity is indicated by the power of the serum to agglutinate cultures of the patient's organisms. By this means, treatment becomes something better than haphazard, and results have so far seemed to be encouraging.

IMPERATORI.

**Prophylactic Mediastinotomy for Perforating Esophageal Foreign Bodies: Report of Three Unusual Cases.**

*Lederer, Francis L., and Fishman, Louis Z. (Chicago), Arch. of Otolaryng., 19:426 (April), 1934.*

An unusual case of multiple small and large sharp foreign bodies within the gastro-intestinal tract is presented. All of these were passed by rectum without complications.

Case histories of esophageal foreign bodies from the literature and from their own experience are presented, the outcome of which has shown that after a diagnosis of esophageal perforation has been made we have no means of determining whether the conservative or radical form of intervention is to be desired.

Safety-pin closers are not always used without danger. The surgeon should be absolutely certain that the point of the safety pin is not embedded, either originally or as a result of purposeful disengagement, before he attempts to make use of such mechanical devices as safety-pin closers.

Prophylactic mediastinotomy is done frequently in Vienna and very infrequently in America. The authors urge its use conservatively.

TOBEY.



**Erysipelas and the Hemolytic Streptococcus in Relation to Otolaryngology.**

McKenzie, Dan. (London), *J. of Laryng. and Otol.*, 49:105 (Feb.), 1934.

Erysipelas is more common in the face and head than in any other region of the body, and most of the cases of postoperative erysipelas, as well as postscarlet fever, occur after ear and nose operations. Although erysipelas is a contagious disease, tending to spread from person to person by direct or indirect contact, observation shows quite clearly that a considerable number of the cases are of autogenous origin. The streptococcus hemolyticus is responsible for a group of clinical happenings which comprise not only many of the acute ear and nasal sinus infections, but also epidemic tonsillopharyngitis, erysipelas, the commoner forms of puerperal septicemia and scarlet fever. Laboratory and clinical experience suggest that it has the remarkable power of varying its behavior to suit its biochemical environment. The writer discusses the different forms that the hemolytic streptococci may assume and suggests that they may spontaneously acquire invasive and toxic properties when they become capable of infecting other people.

Although the streptococcus infection is relatively seldom a direct cause of death, the fact that it leads to so much morbidity is not to be ignored. Every case constitutes a center from which others may be propagated. Efforts in controlling the disease are discussed. It is suggested that cultures to be taken from all acute ears, sinuses and throats and that those with hemolytic streptococcus be treated as infectious. In the hospital wards clean operative cases should be separated from active streptococcal cases.

GOLDSMITH.

**Bronchial and Tracheal Catheterization and Its Clinical Applicability.**

Frenckner, P. (Stockholm). *Supplement Acta Otolaryngologica*, 1934.

This 129-page monograph in English, from Prof. Holmgren's clinic, presents with much detail and thoroughness experimental work with the determination of intrabronchial pressure simultaneously in both lungs, by the use of a double bronchoscope, one tube extending lower than the other.

This instrument carries an inflatable cuff in the trachea, and another, near the end of the longer tube of the double bronchoscope to close off one main stem bronchus. By appropriate connections, manometric readings and drum tracings from both lungs were simultaneously secured; these are especially interesting in cases of pneumothorax and partial atelectasis.

Experimentally it was found possible to maintain forced respiration, in either or both lungs, by use of an accurately constructed



electric pumping device, even when double pneumothorax had been brought about; the exposed lungs filled and emptied regularly, with resumption of normal respiration after closure of the chest wall. Studies were made on gas-collapsed and resection human cases.

The method is of special interest to thoracic surgeons and anesthesiologists. This monograph contains valuable information respecting tracheobronchial physiology, gives due credit to Jackson, Mosher, Elsberg, Meltzer and other American authorities, and contains a useful bibliography.

FENTON.

**Geniculate Ganglionitis (Hunt's Syndrome): Clinical Features and Histopathology.**

Maybaum, J. L., and Druss, J. G. (New York), *Arch. of Otolaryng.*, 19:574 (May), 1934.

The authors report a case in detail with photomicrographs of the ganglion and nerve.

Because of some variations in the clinical manifestations of this syndrome, Hunt considered the following clinical subgroups:

A. Herpes Oticus: This is the simplest expression of the condition, which is probably due to the disease being limited to the geniculate ganglion. It may be preceded by the mild prodromal symptoms of herpes zoster, with preherpetic pains localized in the ear and in the region of the mastoid.

B. Herpes Oticus with Facial Palsy: In addition to the herpes oticus, there is an associated facial palsy on the corresponding side. The paralysis is complete, making its appearance with or soon after the onset of the eruption.

C. Herpes Oticus with Facial Palsy and Hypo-Acousis: In this group herpes oticus and facial palsy are associated with a disturbance in hearing. This disturbance is usually transient and of mild degree. Tinnitus aurium may or may not be present.

D. Herpes Oticus with Facial Palsy and Ménière's Syndrome: This is the severest type. In addition to the symptoms noted in the other groups, the highly disturbing symptoms of Ménière's syndrome, such as tinnitus aurium, deafness, vertigo, nystagmus, vomiting and disturbance of the equilibrium, are also present.

The variations of the syndrome originally described by J. Ramsay Hunt are reviewed. Of additional and unusual interest in the case presented is the histologic evidence of otosclerosis and bilateral labyrinthitis.

TOBEY.

**A Report of Seven Cases of Partial Thoracic Stomach with Short Esophagus.**

Monkhouse, J. P., and Montgomery, S. K. (*London*), *J. of Laryngol. and Otol.*, 48:743 (Nov.), 1933.

This article reports seven cases of partial thoracic stomach with short esophagus of mild degree. In six, the diagnosis is confirmed by esophagoscopy, and in one it rests on X-ray evidence alone. These cases fall into two groups, those with and those without dysphagia. Both types have pain which resembles the flatulent dyspepsia of cholecystitis.

The dysphagia is not steadily progressive as in carcinoma but is intermittent, and for some time, often years, is not severe. It is due to the presence of an ulcerated stricture. Hematemesis can occur in both groups.

The diagnosis rests on X-ray and endoscopic examination. In the former, unless the barium is given in the manner described, the lesion is not seen, and it is essential that the passage of the opaque material should be watched on the screen in order to differentiate from a para-esophageal hernia.

In cases of dysphagia, esophagoscopy shows a stricture, possibly with visible ulceration, and the mucous membrane removed from this level is found to be gastric in character.

In the second group no stricture is seen, but a dilatation may be observed at a level which is certainly above the diaphragm and from which gastric mucosa is obtained.

Dilatation will relieve the symptoms in the obstructive type; those without dysphagia receive some benefit from prolonged medical treatment but do not respond well.

The pathology is discussed, but owing to lack of postmortem material many points are still obscure.

GOLDSMITH.

## NOTICE.

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### THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Cleveland, Ohio, June 11, 1934, during the meeting of the American Medical Association. Sixty-two candidates were examined, of which number fourteen were conditioned or failed.

The Board will hold an examination in Chicago, September 8th, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology, and in San Antonio, Texas, November 16th, during the meeting of the Southern Medical Society. Prospective applicants for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for application blanks.

W. P. WHERRY, M. D., Secretary-Treasurer.

H. P. MOSHER, M. D., President.

## Books Received.

LXXX.

### **The Medical and Orthopedic Management of Chronic Arthritis.**

*Ralph Pemberton, M. S., M.D., F. A. C. P., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Chairman American Committee for the Control of Rheumatism; Member Council on Physical Therapy of the American Medical Association, and Robert B. Osgood, A. B., M. D., F. A. C. S., John Ball and Buckminster Brown, Professor Emeritus of Orthopedic Surgery, Harvard Medical School; Member American Committee for the Control of Rheumatism; Member Council on Physical Therapy, American Medical Association.* Cloth. Octavo of 403 pages with 59 illustrations. New York: The Macmillan Company: 1934. Price \$5.00.

A comprehensive monograph; concise, practical and well illustrated. Of interest to the otolaryngologist chiefly from the standpoint of foci of infection occurring in his terrain. "Among the civilian population beyond early maturity, the teeth constitute the site of most frequent infection. . . . In young individuals the tonsils are the chief offenders. . . . The sinuses less frequently constitute the major focus in the nose and throat, but when they do so operate they may present problems of the first magnitude."

### **Textbook of Physical Therapy.**

*Henrich F. Wolf, M. D., Chief of the Department of Physical Therapy, Mt. Sinai Hospital and Dispensary, New York; President, New York Physical Therapy Society.* Cloth. Octavo of 409 pages with 54 illustrations. New York: D. Appleton-Century Company, Incorporated, 1933.

A good general text. The chapter on Physical Therapy in Otolaryngology, a very sane exposition by Farel Jouard, presents a detailed discussion of the choice and value of various physical agencies in the regional diseases of ear, nose and throat. It is constantly emphasized that physical therapy should be employed as an adjuvant and not as a substitute for medical or surgical therapy. Those conditions which are not suitable for physical therapy are carefully singled out.

### **Otitis Media Aguda En El Lactante (Acute Otitis Media in the Infant).**

*Juan Carlos Oreggia, M. D., Chief of the Service of Otorhinolaryngology, Pedro Visca Hospital, Montevideo.* Paper Quarto of 101 pages, with 37 tables. Montevideo: 1933.

A clinical study based upon a series of 136 histories with numerous charts.

**Nouvelles Consultations Oto-Rhino-Laryngologiques du Praticien (Second Edition). (A Practical Formulary for the General Practitioner.)**

*Georges Portmann, Clinical Professor of Oto-Rhino-Laryngology of the Faculty of Medicine of Bordeaux.* Paper 12mo of 364 pages, with 30 illustrations. Paris: G. Doin and Company: 1934. Price 35 Fr.

The second edition of this manual of prescriptions collected for the use of the practitioner. Many of the formulæ have a distinctly French flavor and have not found a place in American practice.

